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. A. S. O.

THE
EXTRA-OCULAR MUSCLES

A CLINICAL STUDY OF NORMAL AND ABNORMAL OCULAR MOTILITY

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TO

MY STUDENTS

PAST AND PRESENT

WHO HAVE INSPIRED THE WRITING

OF THESE PAGES

PREFACE.

In offering this volume on the ocular muscles to students in ophthalmology, the author is not unmindful of the excellence nor of the abundance of the literature on the subject. The difficulties experienced by students in grasping many of the technicalities, and the enthusiasm which has been apparent each year in their honest efforts to master the subject, have stimulated the writer to attempt to crystallize, and at the same time to simplify, accepted facts, so as to render the course in ocular muscles interesting and practical. The present volume, therefore, is a somewhat amplified presentation of lectures which have been given in the Graduate School of Medicine in the University of Pennsylvania. It is offered to all students of ophthalmology with the hope that it may stimulate greater interest in a most important but often much neglected phase of our specialty. If it is found to be helpful to any who, like the author, experience difficulty in correlating many clinical phenomena, it will have served its purpose.

Because of its clinical character, and the hope that it may be eminently practical, abstruse mathematical problems, which are important but not essential, have been omitted. For similar reasons, theories which have not received general approval are discussed but briefly. Accepted theories do not need special defense. To this end, the author has not felt it necessary to defend his acceptance of the role of the "fusion faculty" in discussing concomitant squint. No theory thus far produced answers so satisfactorily in explaining the clinical phenomena and the methods of correcting the same.

Every effort has been made in the text to give due credit to other authors and to publishers for borrowed diagrams, quotations, and ideas. If the author has failed in any instance to acknowledge the same, it is an unintentional omission. To Professor Whitnall and publishers of "Anatomy of the Human Orbit," the author is especially indebted for many illustrations and for the essential facts, set forth in the chapter on Anatomy.

L. C. P.

Philadelphia, 1927.

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THE EXTRA-OCULAR MUSCLES.

PART I.

ANATOMY AND PHYSIOLOGY.

ANATOMY.[1]

The Orbits. The orbits, or orbital cavities (*fossæ orbitales*), are irregularly cone-shaped bony cavities situated to each side of the upper two-thirds of the nose. The apex of each cone is directed backward toward the sella turcica, while the wide-open end, or base, is directed forward and somewhat outward. The median walls, although uneven in contour, approximate parallelism, while the roof, the floor, and the outer wall incline obliquely from the orbital axes from behind forward. This deviation is especially marked in the outer wall, to a somewhat lesser extent above, and is least marked in the floor. While it is ordinarily described as cone-shaped, the orbit takes on a pyramidal form with a tendency to contraction of both apex and base. The apex, or posterior third, also called the retrobulbar part of the orbit, is narrowed to conform to its needs for housing only the ocular nerve, muscles, bloodvessels, and veins. In this area the pyramid is practically reduced to three sides formed by the roof, the internal and external walls—the latter ceasing at the superior orbital or the sphenoidal fissure. The anterior two-thirds, or bulbar part of the cone, is more or less quadrilateral in shape, the several sides merging into the orbital rim, which is especially well marked in the outer two-thirds of the orbital opening.

---

[1] Inasmuch as anatomy is so strictly technical and exact, the author, with Professor Whitnall's kind permission has followed very closely the anatomical facts as described in the *Anatomy of the Human Orbit.*

Bones of Orbit.—The bones which enter into the formation of these bony cavities are as follows:

1. The roof is formed by the frontal and the lesser wing of the sphenoid.
2. The inner wall is formed by the frontal, lacrimal, ethmoid, and the body of the sphenoid.
3. The floor is formed mostly by the superior maxilla, although the zygomatic and palatine bones complete the structure. In addition, the inferior orbital, or spheno-maxillary fissure, has a share in the formation of the floor, bridged over by the periosteum or periorbita, and the so-called involuntary orbital muscle of Müller.
4. The outer wall is formed anteriorly by the zygomatic bone and posteriorly by the greater wing of the sphenoid and a small tip of the frontal. In its extreme apical end the lateral wall is completed by the superior orbital or sphenoidal fissure—the gap between the greater and lesser wing of the sphenoid. This is bridged by the soft structures which pass through it, by the periorbita, the involuntary orbital muscle of Müller, which extends across its lower extremity from the inferior orbital fissure, and by the annulus of Zinn in its middle third.

Periorbita.—The periosteal lining of the orbital bones is known as the periorbita. It is more or less loosely attached and readily admits of subperiosteal hemorrhage such as may occur in infantile scurvy, in whooping-cough and after trauma, giving rise to spontaneous proptosis of the eyeball and even complete paralysis of one or all of the extra-ocular muscles.

Angles.—The shape of the base is more or less quadrilateral with rounded corners, although many orbits take on an oval contour. It is especially to be noted that the rims or orbital margins are not placed horizontally to the side of the nose, but tend to dip down and out, so that the longer axes, if prolonged, would meet well above the root of the nose. Of even greater importance is the angle formed by the vertical planes or bases of the orbits. Measurements by various investigators in different cases vary from 130 to 150 degrees. It is essential to bear in mind that the outer rim of the orbital cavity is on a plane considerably farther back than the median rim. The advantage of this arrangement lies in the greater freedom from obstruction which the visual line encounters in temporal rotation where a wide field is especially advantageous.

The angle formed by the median and external walls according to Whitnall, who quotes Lagrange, is from 45.9 to 48.6 degrees.

According to the same author, the two median walls are rarely parallel. In most instances the distance between the posterior part of the median walls is several millimeters greater than the

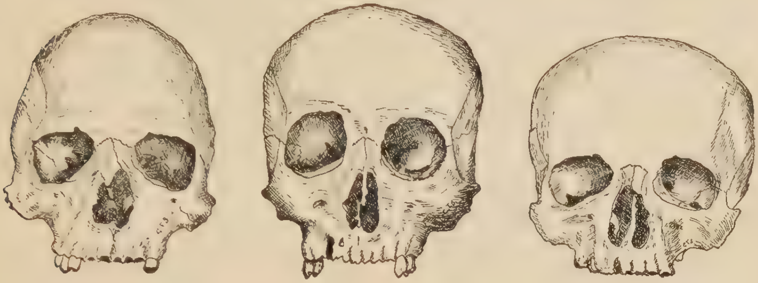

Fig. 1. Fig. 2. Fig. 3.

Figs. 1, 2 and 3.—Characteristic types of orbits. (Stevens' Motor Apparatus of the Eye, courtesy of F. A. Davis Company.)

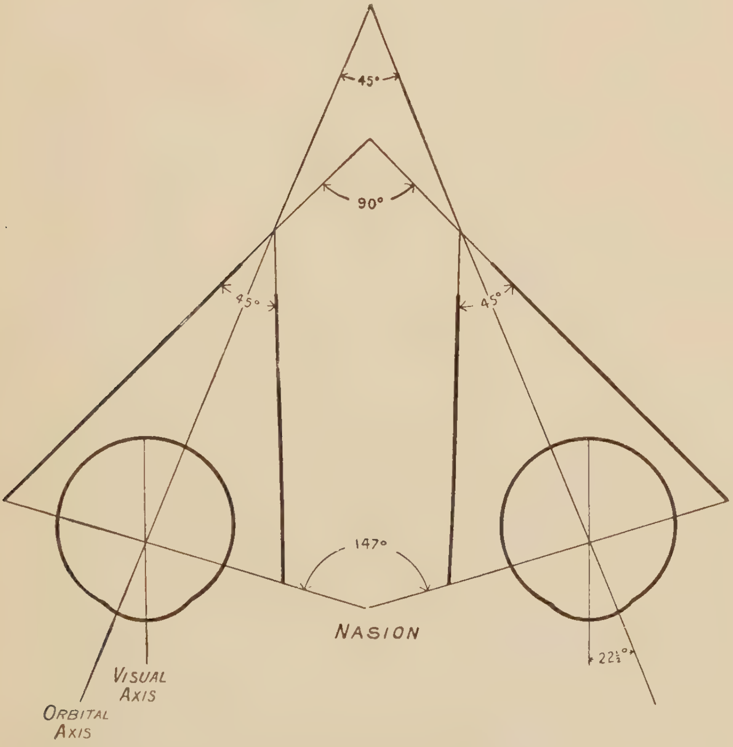

Fig. 4.—Diagram illustrating the angles formed by the orbital walls. (Courtesy of Whitnall and Oxford Medical Publications.)

distance between the anterior ends. The long axis of the orbital cavity is somewhat depressed below the horizontal in adults, but nearly horizontal in children. With the sagittal axis of the skull this axis forms an angle of approximately 23 degrees according to different authorities. If prolonged backward, the two axes should meet slightly posterior to the posterior wall of the sella turcica. It is of interest to note, therefore, that while, the antero-posterior axis of the orbit forms an angle of approximately 23 degrees with the optic axis, it practically coincides with the muscle plane of the superior and inferior recti muscles. This is a fact of great clinical importance.

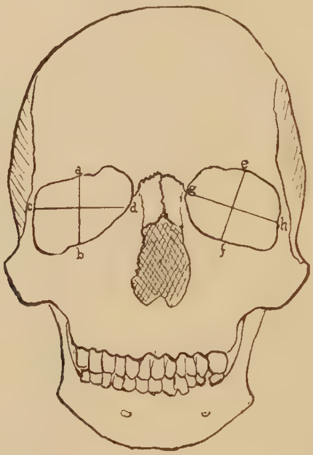

Fig. 5.—Vertical and transverse diameters of the orbit. (Stevens' Motor Apparatus of the Eye, courtesy of F. A. Davis Company.)

Dimensions.—The dimensions of the orbit vary with the age, nationality and race of skulls examined. According to Whitnall, the height of the orbital entrance varies from 30 to 40 mm., while the width is given at from 36 to 50 mm. At birth, the height of the orbit may be equal to the width, but in adult life the width increases, more so in men than in women.

Depth.—The inner wall is shorter than the floor, the roof, or the outer wall. The measurements, which are taken from the apex to the center of the base or to the center of the anterior plane, vary from 39 to 50 mm. In women the depth is less than in men.

Apertures of the Orbit.—The canals and fissures which are of importance for a study of muscles are:

1. The *optic canal* which is situated at the apex of the cone in the lesser wing of the sphenoid. Through this opening pass the

optic nerve, the ophthalmic artery, and the sympathetic fibers from the carotid plexus. The meningeal sheath of the optic nerve, a continuation of the meninges of the brain, forms part of the contents of this canal.

2. The *superior orbital*, or *sphenoidal fissure*, which also forms part of the apex and part of the lateral wall, transmits the third, fourth, sixth, the ophthalmic division of the fifth cranial nerve, the sympathetic root of the ciliary ganglion, and sympathetic fibers from the cavernous plexus, and according to Whitnall, sometimes the sensory root of the ciliary ganglion, the ophthalmic vein and an orbital branch of the middle meningeal artery.

3. The *inferior orbital*, or *spheno-maxillary fissure*, transmitting the infra-orbital nerve and bloodvessels, and the veins connecting the ophthalmic and pterygoid plexuses.

4. The *naso-lacrimal canal* which transmits the naso-lacrimal duct. The latter canal is of interest inasmuch as it opens into the floor of the fossa which houses the lacrimal sac. Near the posterior lacrimal crest of this fossa the inferior oblique muscle takes its origin.

Relational Osteology. The relation of the nasal accessory sinuses to the bony orbit has special significance in the study of certain muscular disturbances in disease of these sinuses. The ethmoidal cells are separated from the orbit by a very thin bony plate. Inflammation of these cells may extend through the bony structure and be the cause of direct involvement of the internal rectus muscle in its course; or rupture through this wall, which is of common occurrence, may directly involve not only the internus, but also the other ocular muscles which are in the immediate vicinity.

Much more intimate, even than the proximity of the ethmoidal cells to the internal rectus muscle, is the relation between disease of the sphenoidal and the posterior ethmoidal cells to the motor nerves of the eye. The sphenoid is located just beneath the optic foramen, and when pneumatization of the lesser wing of the sphenoidal bone occurs, the relation to the third and sixth nerves is quite important. After passing through the sphenoidal fissure, the third and sixth cranial nerves lie in direct contact with the thin layer of bone which separates the sphenoidal sinus from the orbit. An inflammation, therefore, of this cell in many instances gives rise to disturbed function of the muscles supplied by these nerves. Deficient convergence, which has so often been noted, is perhaps the mildest of the forms of disturbed motility observed, although partial palsies and even complete paralyses may be found because of the very intimate anatomical relationship. The fourth nerve is less frequently disturbed because of its slightly more remote position—being located more centrally. Inasmuch, however, as the annulus of Zinn furnishes the common origin of all the external ocular muscles but the inferior oblique, and this ligament surrounds the optic foramen which is directly over the sinus, it is easy to understand how marked general motor disturbance may be associated with chronic or acute sphenoidal disease.

Greenfield Sluder and A. E. Ewing of St. Louis, Mo., have called attention to a relationship between disease of the frontal sinus and the superior oblique muscle.[1] In occlusion of the frontal sinus, Ewing elicited a tenderness on pressure over the trochlear region underneath this sinus which is regarded as characteristic of the headache called "vacuum frontal headache" by these authors. In addition to this sign, when the patient attempts to do close work, headache is increased because of the constant pull of the superior obliques in turning the eyes down and in during the act of reading.

Development.—It is claimed by some that the orbit of a child of six years has practically reached its maturity of growth. Reference, however, to the statistics of Whitnall[2] is convincing that maturity is reached approximately about the age of puberty. The orbital margin of the infant is oval, but as maturity is reached it becomes more angular, due largely to the greater development of the orbital ridges. It is especially noticeable that in men the orbital ridge or margin is heavier and more rounded than that observed in the child or in women.

Whitnall finds considerable variation in the sexes. In women the orbit is more rounded, the orbital ridges are less well defined and, according to Merkel, the female orbit is more elongated. In the male, the prominent ridges furnish protection to the eyeball and harmonize with a more powerfully developed jaw. According to the same author, the walls are larger and more curved because of the heavy rim, and the extreme depth is greater than that found in the female.

While the shape of the orbit may have a direct bearing upon vision, it has not been clearly shown that myopic eyes are found in orbits which differ materially from that of the hyperopic eye. Schmidt Rimpler found about the same orbital index in myopic and hyperopic eyes. Birnbaum (1915), quoted by Whitnall, found that prominent eyes are found to be associated with large and oval ridges, while deep-set eyes are found in orbits with small and rounded openings. Much asymmetry has been observed, and it is more than likely that asymmetry is an important factor in certain forms of motor disturbances of the eye.

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[1] Sluder: Concerning Some Headache and Eye Disorders of Nasal Origin, 1918.

[2] Anatomy of the Human Orbit, p. 99.

Fascia of the Orbit.—Tenon's capsule, also called Bonnet's capsule, *fascia bulbi*, etc., has received much attention by investigators. It probably was known to Galen and to others, but to Tenon of Paris is due the credit of an exhaustive description in 1806. It has been made the subject of original study by many in later years, and this has led students to regard it with increasing complexity rather than simplicity. For practical application, it may be approached advantageously in a more simplified form. As such it can be described as a membrane composed of fibrous and connective tissue which surrounds the eyeball from the cornea to the optic nerve. In addition to this tunic of the eyeball, reflections of the same general character follow the muscles, forming muscle sheaths, while other specially differentiated reflections or thickened portions, known as check ligaments, pass to the bony orbit from the muscle sheaths. A fourth modification of this same fascia is suspended beneath the eyeball with attachments to the outer and inner orbital walls, serving, as it were, as a hammock in which the eyeball rests—the suspensory ligament of Lockwood.

Tenon's Capsule Proper.—The capsule proper, or that portion which surrounds the globe, is pierced by the ocular muscles, blood-vessels, and nerves which pass to the eyeball. Two separate structures have been described by all anatomists, namely, a thin inner membrane and an outer, or the capsule proper. Between these two tissues are spaces which are neither true serous nor yet lymph cavities. The inner or visceral layer is in very close and intimate contact with the globe, firmly attached around the corneal margin but less firmly attached around the optic nerve. In the latter location both structures are so perforated by the posterior ciliary arteries and nerves that the structure is not well defined. At the point of attachment of the muscles the capsule is roughly divided into an anterior and a posterior part. The posterior capsule is much the larger, the ratio being as 2 to 1. It has been pointed out by Merkel and Virchow that the capsule is not continued over the optic nerve to the apex of the orbit, although by others it is claimed that such a covering exists.

The clinical importance of this part of the orbital fascia, or Tenon's capsule proper, is the cavity which is thus formed, in which the eye

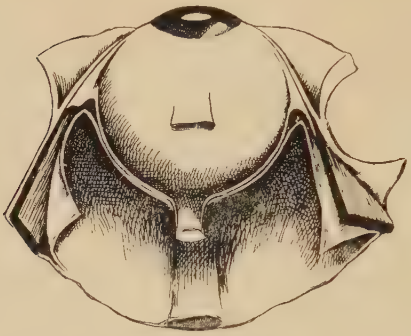


Fig. 6.—Investment of the muscles of the eye, by the capsule of Tenon. Reproduced from Motais's "Anatomie de l'Appareil Moteur de l'Œil" by the kind permission of Professor Motais. (From Stevens' "Motor Apparatus of the Eyes," courtesy of F. A. Davis Company.)

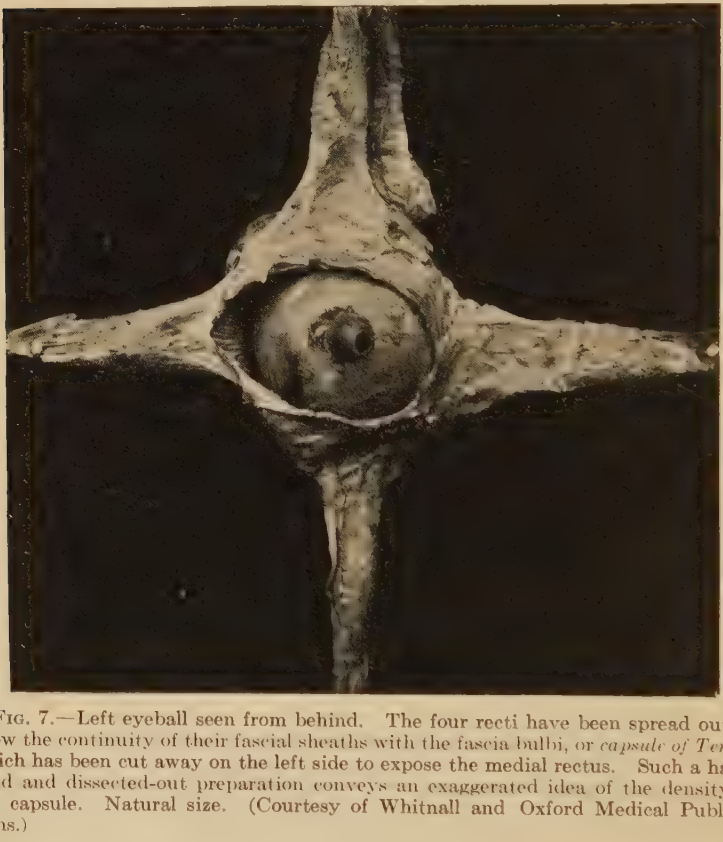
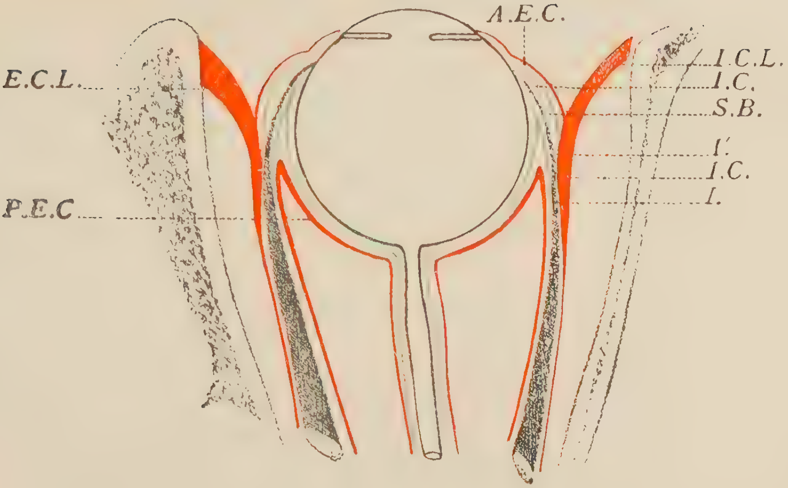


Fig. 7.—Left eyeball seen from behind. The four recti have been spread out to show the continuity of their fascial sheaths with the fascia bulbi, or *capsule of Tenon*, which has been cut away on the left side to expose the medial rectus. Such a hardened and dissected-out preparation conveys an exaggerated idea of the density of the capsule. Natural size. (Courtesy of Whitnall and Oxford Medical Publications.)

PLATE I

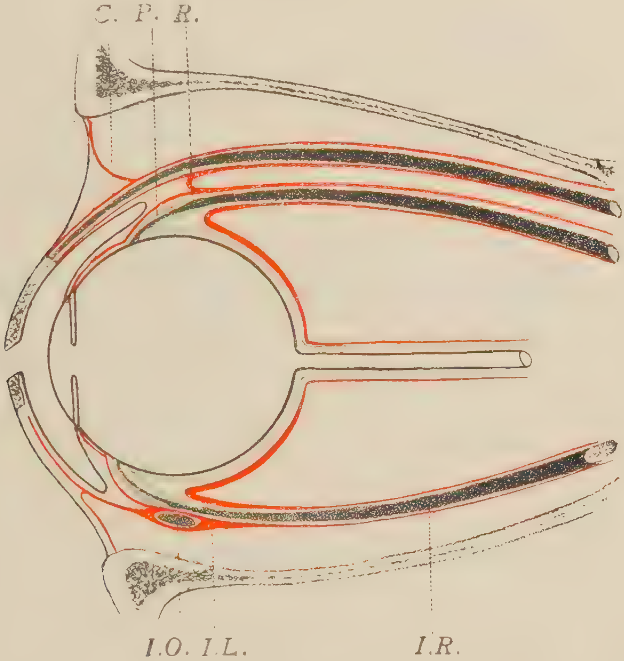
FIG. 1



A Horizontal Section of the Globe and Its Membranes.
(After *Motais*.)

E.C.L. and *I.C.L.* –External and internal check ligaments; *A.E.C.* and *P.E.C.*, anterior and posterior portions of the external capsule; *I.*, *I.'*, *I.C.*, *I.C.*, reflection of internal capsule on the tendon; *S.B.*, serous bursa. (From Maddox's "Ocular Muscles.")

FIG. 2



Vertical Longitudinal Section of Eyeball and Adnexa.
(After *Motais*.)

R, reflection of fascia from the under surface of the levator palpebræ on the upper surface of the superior rectus; *C*, part of the funnel-shaped expansion proceeding to the margin of the orbit; *P*, expansion to contribute to the anterior part of the capsule; *I.R.*, inferior rectus; *I.O.*, inferior oblique; *I.L.*, check ligament of the inferior rectus, embracing the inferior oblique. (From Maddox's "Ocular Muscles.")

rests and from which the orbital fat and other structures are held back and separated. It serves as a socket in a sense, in which the eyeball rotates. The firm union with the eyeball and the intimate relation posteriorly, although not so firm as the anterior attachment, admits of comparatively little movement of the eyeball in the cavity and the capsule for the most part moves with the globe. That a cavity really exists is definitely shown after a careful enucleation when the capsule forms a good wall which holds the orbital structures in place and forms a pocket for the implantation of a foreign body to form a movable stump. Furthermore, by dissection, it is possible to clearly demonstrate the presence of this tunic which can be stripped from the globe with ease although the visceral layer may remain *in situ*.

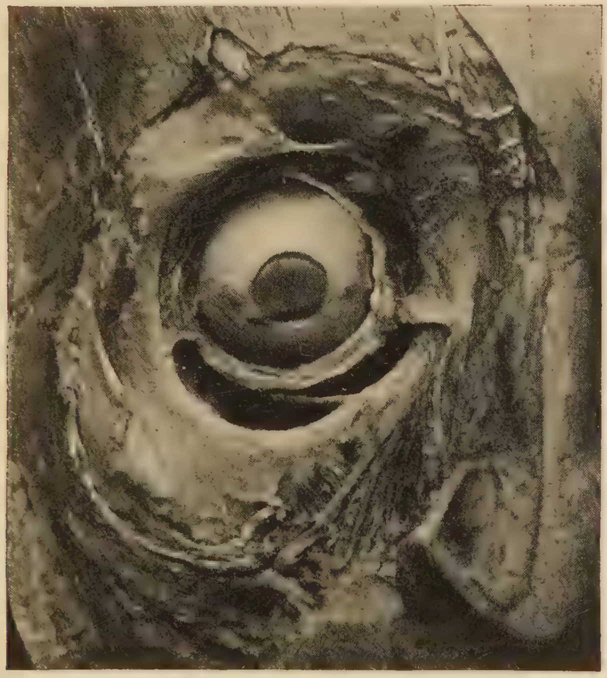


FIG. 8.—The dissection shows the "suspensory ligament" of Lockwood. (Courtesy of Whitnall and Oxford Medical Publications.)

Muscle Sheaths.—The muscle fascia is a reflection of the capsule of Tenon which envelops the muscles from their attachment to the capsule, where it is dense and plainly visible, to their point of origin. This particular phase of the capsule of Tenon makes it difficult to understand. It simplifies matters to regard the muscle sheaths as blending with the capsule proper at the point where the muscles pierce the capsule to become attached to the sclera. Surrounding the globe, there is communication between the several muscle sheaths, a thickening of the capsule proper, so that the eyeball, as it were, rests in a hammock—the so-called suspensory ligament of the eye. This ligament unites with the external and internal check ligaments, and is attached to the bony walls of the orbit, completing the resemblance of this suspensory ligament to a hammock or sling in which the eye rests.

As pointed out by Whitnall the muscle sheaths are firmly attached to the muscles throughout, the surgical importance of which is recognized in tenotomies. The firm union prevents undue retraction of a muscle when severed from its attachment. This limited retraction, however, is also partly due to the restraining action of the check ligament about to be described.

Check Ligaments.—From the sheath of each tendon there are off-shoots or reflections which are attached to the orbital walls, known as check ligaments or ailerons. Two at least are especially well defined, the external and the internal. The external ligament is a broad well-developed band extending from the muscle sheath to the periosteum over the zygomatic tubercle and also to the external canthal ligament. Similarly, the internal ligament extends to the inner wall of the orbit and the internal canthal ligament. Both ligaments send off shoots to the conjunctiva. The superior and inferior ailerons are not so well marked but they are present and can be demonstrated by Howe's method of holding the dissected fascia before a bright light when the thickened tissue is plainly visible.

The clinical importance of the check ligaments rarely receives proper consideration. First, the orbital attachments steady and firmly anchor the eyeball and counteract the backward pull of the four recti muscles; second, the ligaments serve as a check upon the action of the individual muscles. Not only do they limit the overaction of a muscle, but it is more than likely that, being elastic, the action of a muscle, as it nears its maximum contraction, is gradually slowed down and the contraction is smoother than it otherwise would be. That a check ligament actually limits the action of a muscle is easily demonstrated by tenotomy of one of these tendinous attachments, after which the muscle will rotate the eye through a greater arc. Of special interest from a surgical standpoint, is the effect which these ailerons may have upon tenotomies and advancements. After a tenotomy of the internal rectus, for example, the tenotomized muscle retracts but is probably held in place by the check ligament. After the muscle becomes reattached, the arc through which the eyeball can be rotated is much diminished, partly because of its reattachment nearer to the equator of the globe, but more especially because the check ligament already taut, cannot admit of much greater extension of its elastic fibers. As a result, therefore, of tenotomy of the internal rectus there may be permanent insufficiency of converging power and sustained close work is apt to be most uncomfortable and in instances

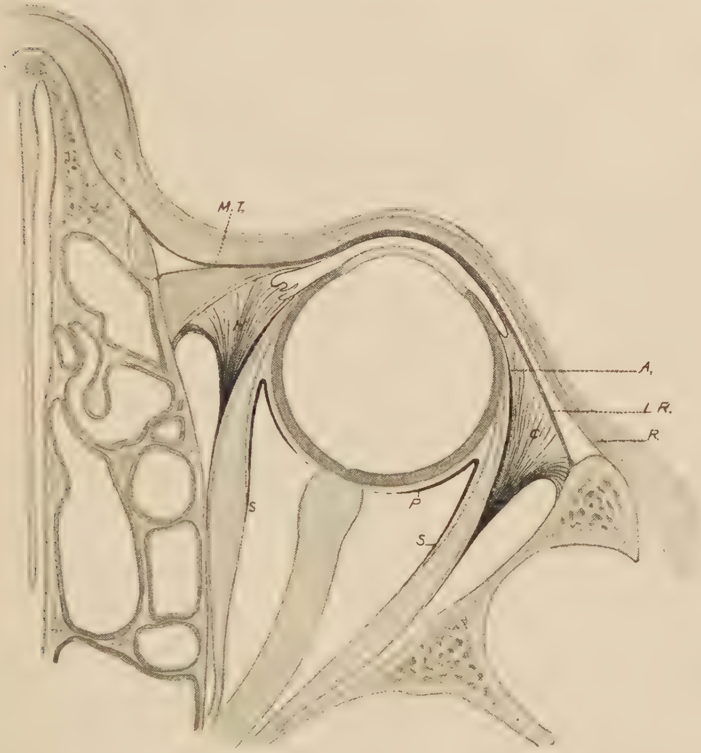


Fig. 9.—Schematic view of a horizontal section through the right orbit to illustrate the *fascia of the orbit*

The *fascia bulbi* or capsule of Tenon is shown by *A*, its anterior part and *P*, its posterior part. The fascial sheaths of the muscles are marked by *S*, and their offshoots form *C*, the lateral and *H*, the medial "check ligaments."

The drawing also illustrates certain points in the anatomy of the eyelids, *M.T.* being the medial palpebral ligament with its two limbs passing in front and behind the fossa for the lacrimal sac, *L.R.* being the lateral palpebral ligament, and *R* the lateral palpebral raphé. (Courtesy of Whitnall and Oxford Medical Publications.)

impossible. The action of the check ligament, after a muscle advancement, aids, or at least admits of an increase in the rotational power of the muscle. This is in marked contrast to the crippling effect produced by a tenotomy. It furnishes the thoughtful ophthalmic surgeon with much evidence in favor of advancements or resections and against tenotomies, a question which is dealt with more fully in the section on the Operative Treatment of Concomitant Squint.

Ocular Muscles.—Six muscles control the movements of the eyeball; the four recti, superior, inferior, external and internal, and the superior and inferior obliques. Although the levator of the lid is not attached to the globe, it is associated with the movements of the superior rectus and will, therefore, be described with the ocular muscles. All of these muscles, with the single exception of the inferior oblique, have a common origin.

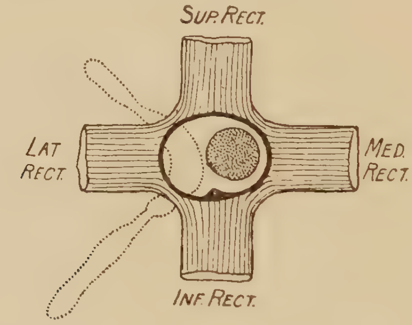


FIG. 10.—Diagram illustrating the origin of the four recti muscles, from the annulus of Zinn, and their relation to the optic foramen and superior orbital fissure. (Courtesy of Whitnall and Oxford Medical Publications.)

The Annulus of Zinn. This circular ligament of Zinn is attached at the extreme apex of the orbit around the optic foramen, above, to the inner side and below, and partly to the outer side; but in addition it spans the superior orbital or sphenoidal fissure and is attached to the anterior bony limits of this fissure. It is oval in its attachment and admits of the passage of nerves and vessels which gain entrance to the orbit through the optic foramen. As pointed out by Whitnall, centrally it is in direct relation to the dura mater as this structure continues to form the covering of the orbital portion of the optic nerve.

The four recti muscles have their origin from the annular ligament closely surrounding the optic nerve. Above and to the outer side but a part of this ligament are the attachments of the elevator of the upper lid and the superior oblique. The structures which pass through this ring-like tendon are the optic nerve and ophthalmic artery embedded in the sheath of the nerve, and branches of the

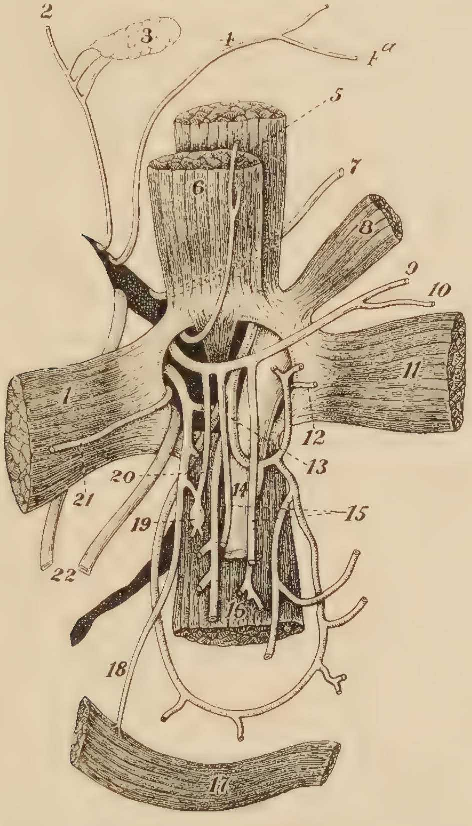


FIG. 11.—*Origin of muscular funnel and the relational anatomy at that position, looking at it from the front.* (1) External rectus showing its single head. (2) Lacrimal branch of the trifacial. (3) Lacrimal gland. (4) Frontal nerve. (4*a*) Supra-trochlear. (5) Superior division of oculomotor nerve. (6) Superior rectus, with levator palpebræ just above it. (7) Trochlear nerve. (8) Superior oblique. (9) Nasociliary. (10) Infratrochlear. (11) Internal rectus. (12) Branch of oculomotor to rectus medialis. (13) Ophthalmic artery. (14) Optic nerve. (15) Long ciliary nerves. (16) Inferior rectus. (17) Inferior oblique. (18) Nerve to inferior oblique. (19) Ciliary ganglion. (20) Long ciliary branch. (21) Abducens nerve. (22) Ophthalmic vein, if in a single root, it generally emerges through the muscular funnel. (After Goldnamer's "Anatomy of Eye and Orbit.")

carotid plexus of the sympathetic. To the outer side of the optic nerve are the structures which pass through the sphenoidal fissure, the third or oculo-motor nerve, the sixth or abducens, the nasociliary branch of the fifth or trigeminal, and usually the superior ophthalmic vein. It is to be noted that the fourth or trochlear nerve is the only one of the ocular nerves which does not gain entrance to the orbit through the annulus of Zinn passing through the sphenoidal fissure externally to the ligament. All the structures enumerated are compactly gathered together in this comparatively small space and are in intimate relation with the origin of the muscle cone and the muscles themselves in the early part of their course.

Superior Rectus.—In common with the other muscles, the superior rectus has a narrow tendinous origin from the annulus of Zinn, an elongated fleshy belly of muscle tissue which terminates in a thin tendinous expansion which is inserted into the sclera about 8 mm. posterior to the sclero-corneal junction and in front of the equator of the eyeball. Its tendon is 5.8 mm. long and 10.6 mm. in breadth. Its total length is 41.8 mm. In the beginning of its course it is intimately associated, in the same muscle sheath, with the *levator palpebræ superioris* and is closely associated with the movements of this muscle. It is the longest and weakest of the recti muscles. When the eyeball is in primary position it acts as an elevator because of its attachment in front of the equator. As its general direction forms an angle of 23 degrees with the antero-posterior axis of the eye, it also rotates the eye to the nasal side and tilts the upper end of the corneal meridian nasalward (intorsion). It is supplied by a branch of the third nerve.

Inferior Rectus.—The inferior rectus has its origin from the inferior part of the annulus of Zinn, passes forward and outward in the same general direction as the superior rectus, hugs the equator of the eyeball and is inserted, by a tendinous expansion, 5.5 mm. long and 9.8 mm. broad, into the sclera 6 mm. posterior to the inferior sclero-corneal junction. Its total length is 40 mm. Like the attachment of the superior rectus, the convexity is forward and the center of the tendon is slightly to the nasal side of the vertical meridian of the eyeball. It turns the eyeball down and in and rotates the upper end of the corneal meridian templeward (extorsion) Through fibrous bands it is attached to the conjunctiva of the cul-de-sac and the inferior cartilaginous plate of the lower lid. Through these attachments it bears a similar relation to the lower lid that the superior rectus does to the upper. It is supplied by a branch of the third nerve. The muscle planes of the superior and inferior recti are practically the same, forming an angle of 23 degrees with the geometric axis of the eyeball. To be absolutely accurate, there is a very slight difference in the direction of the muscle planes of these

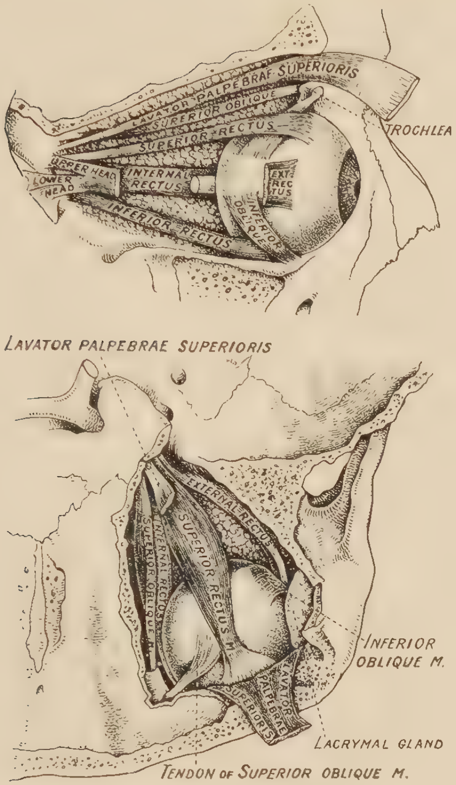


Fig. 12.—Diagrams in which the ocular muscles are viewed laterally and from above.

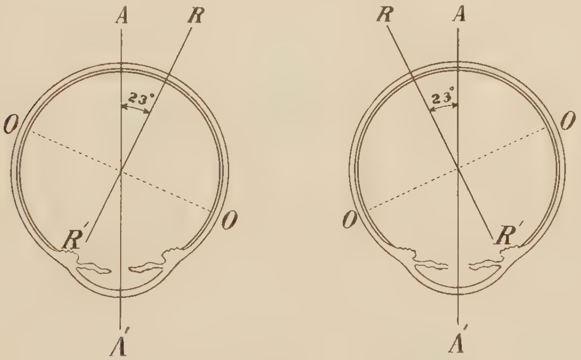


Fig. 13.—Diagram showing the muscle planes of the superior and inferior recti in relation to the antero-posterior axes of the eyes.

two muscles, but for practical purposes and in order to avoid confusion, they may be considered as identical. In combined and equal action of these muscles, therefore, the vertical pull and the forces which tilt or rotate the cornea are neutralized and the resulting deviation is that of internal rotation.

Internal Rectus.—The internus has its origin from the inner side of the ligament of Zinn. It passes forward parallel and close to the inner wall of the orbit to the equator of the eyeball, remaining in contact with the globe to its attachment 5.5 mm. from the sclerocorneal margin. The tendon is 10.3 mm. broad and 3.7 mm. long. Its length is 40.8 mm. It is the heaviest, thickest, and most powerful of the four recti muscles. It is supplied by a branch of the third nerve. It rotates the eye inward and, when normally attached, has no torsional action.

External Rectus.—The external rectus is said to have a double origin, from the ligament of Zinn proper and the lateral wall of the sphenoidal fissure. It, however, virtually has but one attachment, namely, from the outer side of the ligament of Zinn where this tendon spans the sphenoidal fissure. The muscle passes forward and outward close to the outer wall of the orbit to the equator of the eyeball and is attached to the sclera by a tendon 8.8 mm. in length and 9.2 mm. in width, about 7 mm. from the outer limbus. The length of the entire muscle is 40.6 mm. It is supplied by the abducens or sixth nerve. It rotates the eye to the temporal side and has no torsional action.

It will be noted that the four recti muscles are approximately 40 mm. in length. The internal rectus is the heaviest and thickest in its fleshy part; the external rectus is second and inferior rectus third; the superior is the lightest in muscle structure.

In the length and breadth of the four recti tendons and the exact point of attachment to the sclera, there is much variation. As given above, the distances from the cornea are, in round numbers, 5.5, 6, 7 and 8 mm. for the internal, inferior, external and superior respectively. The exact figures of Fuch's which are pretty generally accepted are 5.5 mm., 6.5 mm., 6.9 mm., and 7.7 mm. For practical purposes the figures 5, 6, 7 and 8 are easily remembered. In tendon breadth the measurements of Fuch are also accepted, 10.3 mm., 9.8 mm., 9.2 mm. and 10.6 mm in the order as given above. Merkel's measurements of the length of the tendons are 3.7 mm., 5.5 mm., 8.8 mm. and 5.8 mm.

In the length of the superior and inferior tendons practically all agree. There has crept into text-books, however, much variance in the lengths of the internal and external recti tendons. As this has a distinct bearing upon advancements, resections and tendon transplantations for the correction of concomitant and organic

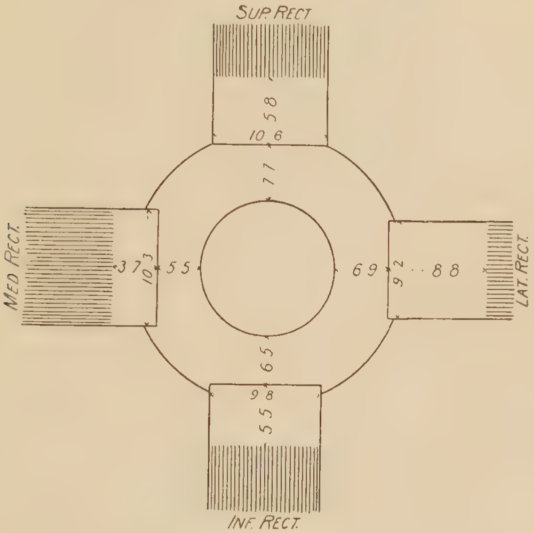


FIG. 14.—Spiral of insertions of the recti muscles. (Courtesy of Whitnall and Oxford Medical Publications.)

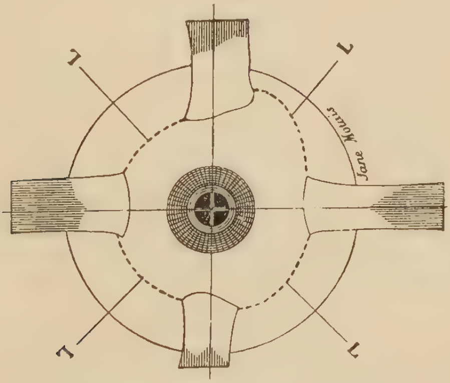


FIG. 15.—Insertions of the recti muscles according to Matais. (Courtesy of Whitnall and Oxford Medical Publications.)

squint, this error should be corrected. The external rectus has the longer tendon, 8.8 mm., whereas the internal rectus has a tendon of 3.7 mm. in length.

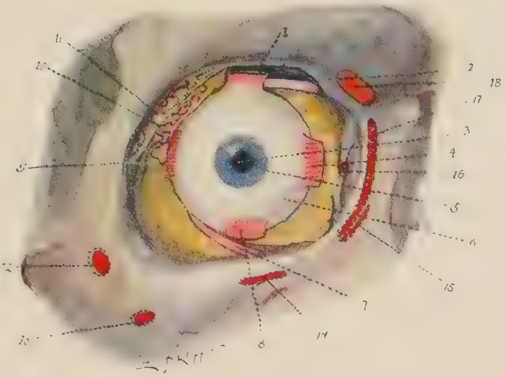
It should, also, be noted that the insertions of the superior and inferior recti vary some from the insertion of the lateral recti. The attachments toward the nasal side are further forward than the temporal edges, as shown in the accompanying diagram.

In addition to the visible line of attachments, as shown in Fig. 15 not infrequently tendinous fibers may be found to be adherent to the sclera as much as 5 mm. posteriorly. In muscle advancement operations, when the tendon is completely detached these fiber bands are frequently encountered. They are factors with which the surgeon must reckon. Defective rotations are encountered when rotational ability, for some reason, is less than would seem to be indicated by the line of the regular tendon attachment. The pull of the muscle, as generally accepted, is from the posterior part of adherence to the sphere. Such attachments in tenotomies, if disregarded, may interfere with a maximum result and, on the other hand, they may save the surgeon from obtaining embarrassing results in the too frequent practice of this questionable operation, by preventing too great a retraction of a tenotomized muscle.

The Superior Oblique.—The superior oblique arises from a slender tendon to the inner side of the optic foramen, from which it is separated by the internal rectus, from the angle between the ligament of Zinn and the orbital periosteum. According to Whitnall, it may also be attached for some distance to the inner wall of the orbit. The muscle belly is more rounded than the other muscles, passes forward in the angle formed by the superior and medial orbital walls. It follows closely the nasal wall of the ethmoids above the internal rectus. Just before it reaches the orbital margin it becomes tendinous and passes through a cartilaginous ring known as the *trochlea.* After passing this ring it turns abruptly backward forming an angle of about 54 degrees with its muscular course, and continues outward and slightly downward, expanding into a flattened tendon which passes under the superior rectus to be attached to the sclera on the outer and upper aspect of the globe. The total length of the tendon is 20 mm. and its breadth at the point of attachment, according to Fuchs, is 10.7 mm. By Fuchs and others the width is found to vary. The distance from the cornea to its anterior edge has been found by various investigators to vary from 14 to 17.9 mm. Its posterior edge is approximately 7.5 mm. from the optic nerve. The distance between the outer edge of the superior rectus and the anterior edge of the superior oblique, Whitnall found to vary from 0.5 to 8 mm. The two tendons, however, are occasionally found to be connected by strands of connective tissue.

PLATE II

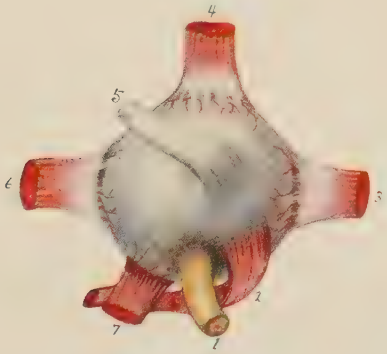
FIG. 1



Anterior View with Muscular Connections in and Around Orbit.

(1) M. superior rectus; (2) tendon m. superior oblique; (3) pupil; (4) m. medial rectus; (5) iris; (6) sclera; (7) m. inferior oblique; (8) m. inferior rectus; (9) m. lateral rectus; (10) lateral horn of m. levator palpebræ superior; (11) lacrimal gland showing the two lobes; (12 and 13) origin zygomaticus muscle; (14) origin infra-orbital muscle; (15) origin levator anguli oris muscle; (16) insertion pars m. lacrimalis (Horner's muscle); (17) origin orbicularis oculi muscle; (18) origin corrugator supercilii muscle. (From Goldnamer, "Anatomy of the Eye and Orbit." Courtesy of The Professional Press, Inc.)

FIG. 2



The Eyeball from the Rear.

Showing the insertions of the muscles and their relations to each other and to the optic nerve, also some secondary insertions are indicated. (1) Optic nerve; (2) inferior oblique; (3) external rectus; (4) superior rectus; (5) superior oblique; (6) internal rectus; (7) inferior rectus. (From Goldnamer, "Anatomy of the Eye and Orbit." Courtesy of The Professional Press, Inc.)

It is supplied by the fourth or trochlear nerve which enters the substance of the muscle about 12 mm. from its origin.

The *trochlea* or pulley consists of cartilage, 4 by 6 mm. (Whitnall) attached by its edges to the frontal bone by fibrous bands forming a ring through which the tendon plays. The tendon is invested with a fascial sheath which is attached to and ends at the trochlea. The trochlea has an endothelial lining but not a true synovial membrane (Whitnall and Macalister).

The superior oblique rotates the eyeball down and out and causes the upper end of the corneal meridian to be rotated inward.

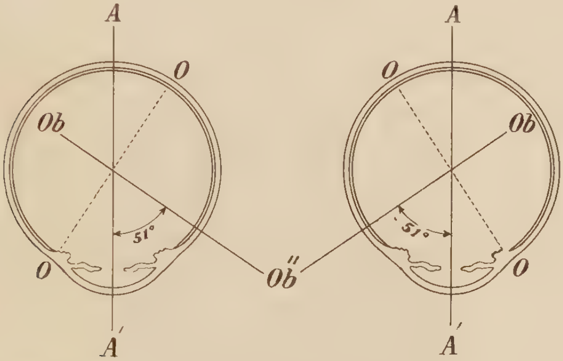


Fig. 16.—Diagram showing the planes of the oblique muscles in relation to the antero-posterior axes of the eyes.

The Inferior Oblique.—The inferior oblique has its origin at the anterior inferior angle of the orbit, from a shallow depression on the orbital floor, close to the outer side of the lachrymal fossa which houses the lachrymal gland. The muscle, somewhat flattened, passes backward underneath the inferior rectus to which it is quite intimately attached by fibrous bands, and is inserted into the posterior hemisphere of the eyeball by a short tendon 2.6 mm. long. The line of its insertion is in the posterior hemisphere making an angle of 25 degrees with that of the superior oblique. Its posterior border is 5.2 mm. from the optic nerve in hyperopic eyes and 7.1 mm. in myopic eyes (Whitnall). The distance of its posterior margin from the position corresponding to the fovea is 2.2 mm. Fuchs found the anterior margin from 17.3 to 19.1 mm. from the cornea. The anterior margins of the obliques are separated about 15 mm. while the posterior margins are 11.9 mm. apart.

It is supplied by a branch of the third nerve which enters the muscle about the middle of its posterior border.

From eyes front, the inferior oblique elevates the eye, turns it out and rotates the upper end of the corneal meridian out (extorsion).

In comparing the action of the two obliques, it is best to disregard the muscular part of the superior oblique and to speak of its action as though it had its origin at the *trochlea*. With this method of orientation it will be noted that the two muscles have the same general direction. The inferior oblique, however, is attached farther back than the superior oblique and the origin of the inferior oblique is father forward than that of the superior. The muscle planes, therefore, do not accurately coincide, but for practical and clinical purposes their muscle planes are regarded as about the same, making an angle of about 51 degrees with the optic axis. They are direct opponents in their upward and downward pull and in torsion, the superior oblique being an intorter of the vertical corneal meridian and the inferior an extorter. In lateral action they are both external rotators. In synchronized action, therefore, the vertical pull and torsion are neutralized and their combined action results in temporal rotation.

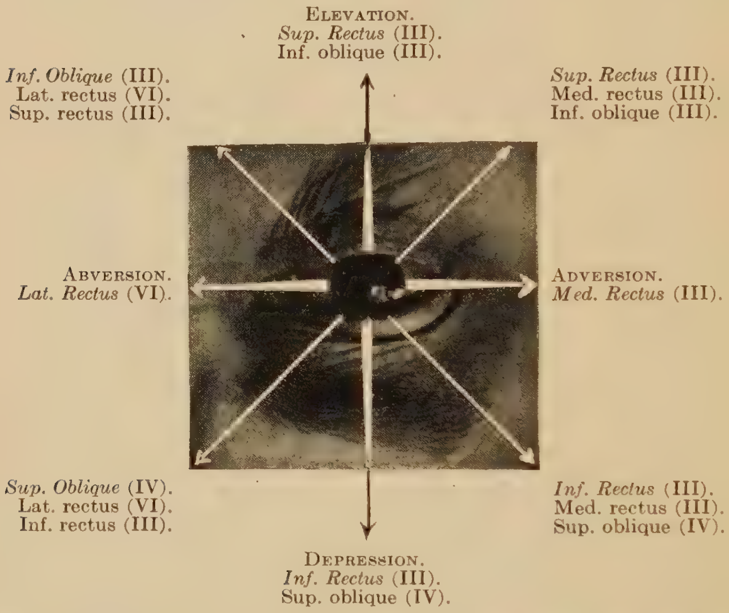


FIG. 17.—Scheme of movements of the cornea, with the muscles that affect them. The predominant muscles are in capital letters. The Roman numerals after the names indicate the nerve supply. (Courtesy of Whitnall and Oxford Medical Publications.)

Levator Palpebræ Superioris.—The elevator of the upper lid, although strictly speaking not one of the muscles which move the eyeballs, is so inseparably associated with the action of the superior rectus and its synergists, and at the same time is so intimate in its course, insertions and associations, that its description by Professor Whitnall will be closely followed. The muscle arises by a slender tendon from the lesser wing of the sphenoid to the outer side and above the optic foramen, being separated from the foramen by the superior rectus tendon, with which it is blended. As it expands into the muscle proper it passes forward and outward, in close contact with the superior rectus beneath, the fascial sheaths of the two muscles being intimately blended throughout the greater part of the muscles, and above it closely hugs the roof of the orbit to the orbital margin. The inner edges of the superior rectus and the levator are in the same plane, but the outer edge of the superior rectus extends considerably to the temporal side in the forward part of the orbit.

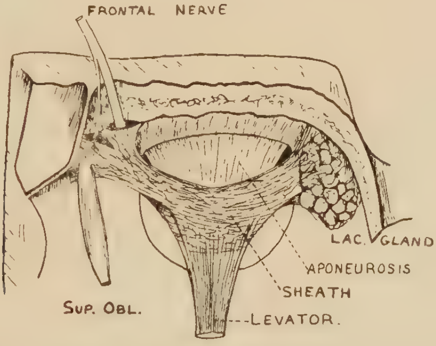


FIG. 18.—Dissection of the levator palpebræ superioris from above in right orbit. The muscle is shown drawn backward and downward to make tense the *transverse ligament* formed by its sheath. The lateral attachments of this ligament are seen on the medial side to the bone below the superior oblique muscle, to its pulley, and by a thin slip of fascia to the margins of the supra-orbital notch; on the lateral side to the stroma of the lacrimal gland and also by direct insertion to the bone. (Courtesy of Whitnall and Oxford Medical Publications.)

Attachments.—The muscle has a three-fold attachment. The palpebral aponeurosis which is the expanded tendon of the muscle spreads at the margin of the orbit and descends over the glove on either side, in arch-like expansions, the outer and inner "horns" which are attached to the outer and inner angles of the bony orbit (Fig. 18). The central or main aponeurotic expansion is attached subcutaneously throughout the free border of the lid, the fibers passing anterior to the cartilaginous plate. The length of the aponeurosis is 7 mm. and the thickness about 0.5 mm. It is of importance to emphasize the fact that the tendinous or main attachment of the muscle is cutaneous and precartilaginous. By these two attachments, the cutaneous and the osseous, two spans or cavities are created, the "pretarsal" which lies between the cartilage and the aponeurosis, and the "preseptal," which lies between the aponeurosis and the *septum orbitale* and the fatty cushion of the eyebrow above.

The osseous portions of the attachments are equally interesting and of clinical importance. The inner fibers are attached to the bony orbit. In its course, its fibers are in contact with the reflected tendon of the superior oblique muscle, finally reaching the internal palpebral ligament. The outer "horn" is better developed, "cuts into the tarsal gland" and below the gland is inserted into the orbital tubercle of the zygomatic bone.

The third attachment is that made with Müller's muscle, the involuntary muscle of the lid. These fibers were described by Müller in 1859.[1] The muscle consists of red fibers about 10 mm. in width antero-posteriorly which have their origin in elastic tendons from between the inferior part of the striated structures of the muscle and are attached to the upper edge of the tarsal plate by similar elastic fibers. Less well developed, but demonstrable, are similar fibers in the lower lid. These fibers are supplied by the sympathetic system, probably, as suggested by Whitnall, from the cavernous plexus. At all events, it enters into an important syndrome which is caused by cervical sympathetic irritation or destruction, a widened palpebral fissure in the former and ptosis of a moderate degree in the latter instance.

In addition to the three-fold attachment of this muscle there is an attachment between the fascial sheath of the levator and the superior retro-tarsal folds of the conjunctiva or fornix.

The levator is supplied by the branch of the third nerve which is first distributed to the superior rectus muscle. This adds another link to the synchronous action of these two muscles.

The *levator palpebræ superioris* elevates the upper lid and because of the connection of its fascial sheath with the superior conjunctival fornix, the conjunctiva is elevated at the same time. It is probable that the greater development of the outer osseous "horn" attachment, causes greater restriction of upward movement of the lid in its outer part, and accounts for the freer movement of the lid in its inner half.

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[1] Verhandlung an der P. M. G. in Würzburg, 1859, 9, 244.

In our clinical observations of the synchronous upward movement of the eyeball, the upper lid and the retro-tarsal conjunctiva, one cannot fail to be impressed with the anatomical arrangement which makes it possible. It also points the way toward the more frequent practice of the operation of Matais in selected types of paralytic ptosis.

Abnormalities.—Abnormalities in the ocular muscles are not rare. Many undoubtedly go unrecognized in the operating and the anatomical rooms in dissections. They are due for the most part to improper cleavage of the mesoplastic tissues. Abnormal origins, abnormal distributions, total absence and imperfect developments are recorded. Unusual forms of muscle imbalance may be due to one or more of these defects. Congenital types of paralytic squint are frequently due, either to a poorly developed or to a totally absent muscle. These cases as a rule are accompanied by a marked degree of facial asymmetry and unusual head postures. In other instances, careful dissections in the dissecting room may uncover abnormal off-shoots or supernumerary muscles which may or may not have played a role in some unusual muscular disturbance during life.

As might be expected from the complicated character of the course and distributions of the attachments of the *levator palpebræ superioris,* many abnormalities have been described. For the more minute study of these interesting abnormalities, the reader is referred to Whitnall on the *Anatomy of the Human Orbit* and to other works on the anatomy of the eye.

Blood Supply.—The source of the blood supply to the muscles, in common with most of the eye structures, is the ophthalmic artery, a branch of the internal carotid. The muscular branches are, as a rule, two, which divide quite freely to supply the muscles. The inferior branch supplies the internal rectus, the inferior rectus, and the inferior oblique. The superior trunk sends branches to the superior rectus and the levator, to the external rectus and the superior oblique. These muscular branches finally become the anterior ciliary arteries. With the exception of the external rectus, which has but one ciliary branch, the recti muscles are accompanied in the anterior part of their course by two ciliary branches which penetrate the sclera about the point of attachment of the recti muscles to the sclera. This fact is worth remembering because in tenotomies there usually is quite free bleeding when the tendon is severed.

Veins.—The blood from the eyeball and its *adnexa* is carried off by the ophthalmic vein which empties into the cavernous sinus, second by a free anastomosis of the superior ophthalmic and the angular vein, and third, through the inferior orbital fissure into the pterygoid plexus (Whitnall).

Clinically the venous course of the blood is of importance insofar as the extra-ocular muscles of the eye are concerned, largely in connection with the pathological processes in the cavernous sinus through which the motor nerves of the ocular muscles pass.

Nerves.[1]—The motor nerves of the ocular muscles, including the levator of the upper lid, are the third or oculo-motor, the fourth or trochlear, and the sixth or abducens. In order to thoroughly appreciate the complex symptomatology of ocular muscle palsies as well as the innervations which originate in the brain cortex, it will be necessary to follow most minutely each nerve from its nuclear source, through the fasciculi which make up of the nerve trunk proper; its exit from the brain, its intracranial course, through the cavernous sinus, through the supra-orbital fissure, through the orbit and finally its distribution. While this method of study may seem to lead us far into the field of anatomy and perhaps into neurology, a shorter and more direct method of approach can only lead to difficulties in accounting for and explaining many obscure forms of palsies of the ocular muscles.

Oculo-motor Nerve.—The third cranial nerve, also called the oculo-motor, has a nuclear origin which is as complex as its wide distribution to the superior, inferior, and internal recti, the inferior oblique and levator of the upper lid as well as branches through the ciliary ganglion which supply the ciliary muscle, and the sphincter of the pupil. This wide distribution, giving rise to a varied symptomatology in disease, is simplified by the multiple nuclear centers which together are known as the nucleus of the third nerve.

The nucleus lies in the upper part of the cerebral peduncle, just beneath the floor of the aqueduct of Sylvius, extending about 5 mm. from the posterior part of the third ventricle to the anterior end of the third ventricle. In its lower part it is in close relation with the nucleus of the fourth or trochlear nerve. The right and left nuclei of the third nerve are somewhat separated above but they are in closer relation below, where each also is in contact with the median longitudinal bundle of the corresponding side (Fig. 19). The nuclear mass consists of a larger lateral collection of centers which supply the extrinsic muscles referred to above and two more centrally located and smaller masses which are destined to supply the intrinsic muscles of the eye. The upper or anterior center of this latter group is known as the Edinger-Westphal nucleus. The inferior intrinsic nucleus is centrally located and is found with its fellow nucleus from the other side. Investigators are not thoroughly in accord as to the exact serial position of the lateral centers which supply the extrinsic muscles. The disagreement, arising from a lack of accurate knowledge on this anatomical phase, is due to the fact that experimental studies can only be made on lower animals and we have no assurance that the same anatomical arrangement found in them applies to man. The schematic drawing given below, after Bernheimer, is the arrangement usually accepted.

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[1] Closely following Professor Whitnall's text.

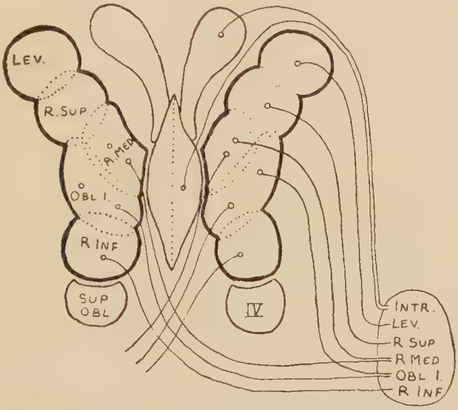

FIG. 19.—Scheme representing the constitution of the oculomotor nucleus, modified after Bernheimer (1904). The nuclei of both sides are shown. (Courtesy of Whitnall and Oxford Medical Publications.)

The determination of the intercommunication between corresponding nuclei on each side is also attended with difficulties. The majority of the nerve fibers from the nuclei pass out on the same side on which they originate but there is some communication at least between the lateral nuclei of the inferior recti and the internal recti. It is generally supposed that the fibers from the nuclei of the superior recti and the levators pass directly without communication; that the internal recti have direct and communicating fibers while the fibers of the inferior recti are all communicating or crossed.

Communication with other structures in the brain generally accepted are: (1) With the anterior corpora quadrigemina; (2)

with the motor centers in the cortex—the conjugate centers for eye movements; (3) through the superior cerebellar peduncles with the cerebellum; (4) with the trochlear, abducens and vestibular nerves. Some of these communications in the latter group are probably through the median longitudinal bundle with which it is in contact in the lower part of the nucleus. The synchronous action of the external of one side and the internal rectus of the other presupposes that there is communication between the third and sixth nuclei, probably also through the same median longitudinal bundle. In addition, fibers from the third nerve nucleus apparently "enter the upper facial nucleus, and take part in the innervation of the orbicularis palpebrarum" (Bing). This fact is established, according to the same author: (1) By "paresis of the orbicularis which accompanies nuclear lesions of the oculo-motor nerve;" and (2) by "the upward turning of the eyeball which takes place simultaneously with closure of the lids."

The fasciculi arising from the nuclei on the same side and from some of the crossed fibers from the nuclei on the opposite side, pass through the median longitudinal bundle, cross the tegmentum of the pons, the red nucleus and emerge on the inner side of the cerebral peduncle to form the nerve trunk proper.

The trunk has its origin in the interpeduncular space above or anterior to the pons, usually by two branches or fasciculi separated by a small branch of the posterior cerebral artery. In the intracranial part of its course to where it passes through the dura to enter the cavernous sinus, it has a length of about 25 mm. It is closely invested by the pial membrane and lies between the posterior cerebral and the superior cerebellar arteries, a clinical fact of much importance.

As it enters the cavernous sinus, after piercing the dura, it lies in contact with the anterior clinoid process of the skull. In the cavernous sinus it is buried in the lateral wall above the trochlear nerve but beneath this nerve as it makes its exit from the sinus to enter the orbit through the superior orbital fissure. At this point, it divides into a superior and inferior portion, each of which passes through the ligament of Zinn within the muscle cone. Along with the abducens nerve and other structures it is compactly crowded together in this narrow muscle cone, about 3 mm. in diameter.

In the orbit the branches separate; the superior or smaller branch passing upward over the inner edge of the optic nerve to enter the superior rectus muscle. After supplying this, it passes on to supply the *levator palpebræ superioris* which lies above the superior rectus. The inferior or larger branch, after supplying the internal and inferior recti, passes forward beneath the optic nerve to supply the inferior oblique. The branch which terminates in the inferior oblique, sends a strong motor root to the ciliary ganglion.

From an anatomical standpoint paralysis of the oculo-motor nerve has special interest. In addition to the cause of its paralysis, in which any or all of the ocular muscles may share, certain anatomical relations have a special interest. Aneurysm of the posterior cerebral or superior cerebellar arteries may cause paralysis because of its position in the angle formed by these two vessels. Similarly the posterior communicating artery, which connects the posterior cerebral with the internal carotid in establishing the circle of Willis, may cause compression. Aneurysm of the internal carotid has been accompanied by oculo-motor palsy. Whitnall sees a possible explanation of cases of recurrent oculo-motor palsy in vascular spasm or paralysis of the vaso-motor nerves of these vessels which may cause distention of the vessels and secondarily compression of the nerve.

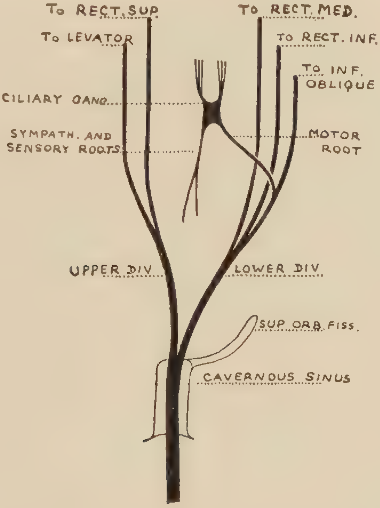

FIG. 20.—Diagram of the oculo-motor nerve, with its branches. (Courtesy of Whitnall and Oxford Medical Publications.)

Its intimate relation to the anterior clinoid process makes it vulnerable to paralysis in fractures of the base, while its relation to the sphenoidal sinus when the sinus extends under the optic foramen, renders it especially susceptible to disturbed function in disease of this sinus.

The Trochlear or Fourth Nerve.—The trochlear is the motor nerve which supplies the superior oblique muscle. It takes its origin beneath the floor of the aqueduct of Sylvius opposite the upper part of the inferior colliculus or anterior corpus quadrigeminum. It is close to, but detached from, the lower part of the gray matter which constitutes the oculo-motor nucleus. The fibers from the nucleus run laterally and downward, decussate in part with corresponding fibers of the nucleus of the opposite side, emerge from the brain just below the inferior colliculus and wind around the cerebral peduncle, immediately above the pons. In this part of its course, after emerging, it lies within the subarachnoid space, and runs

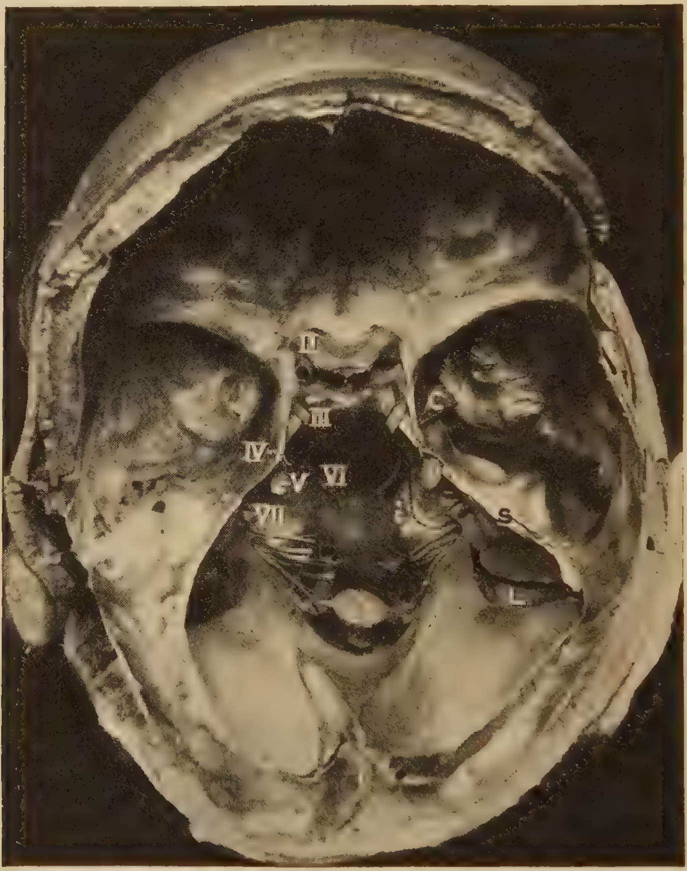

Fig. 21.—The base of the skull with dura *in situ*, showing *the nerve exits and position of sinuses*. (Courtesy of Whitnall and Oxford Medical Publications.)

between the posterior cerebral and superior cerebellar arteries. It pierces the dura mater behind the third nerve and the posterior clinoid process (Fig. 21), and enters the cavernous sinus. It is beneath the oculo-motor nerve in the first part of its course in the lateral wall of the sinus, crosses and emerges from the sinus above the third nerve. It enters the orbit through the lateral part of the superior orbital fissure, being the only motor nerve which does not enter the orbit through the annulus of Zinn and the muscle cone. Its course in the orbit is short, crossing the superior rectus obliquely, passing forward and internally, immediately beneath the periosteum of the orbital roof. It is distributed to the superior oblique muscle.

The intracranial course of the nerve is 40 mm. (Whitnall). Its close relation to the third or oculo-motor nerve renders it vulnerable to the same pathology to which the third nerve is subject.

The Abducens or Sixth Nerve.—The sixth or abducens nerve, like the trochlear supplies but one ocular muscle—namely, the external rectus.

The nucleus lies beneath the *eminentia teres* of the fourth ventricle. It lies centrally within the loop of the facial nerve. Its fibers pass forward to the lateral side of the pyramidal tract after traversing the entire thickness of the pons and emerge at its lower border. The fibers do not decussate but are direct.

The central connections of the nucleus of the abducens nerve have important applications. These connections are principally with the visual and motor cortical areas, with the nuclei of the third, fourth and vestibular nerves through the medial longitudinal fasciculus. A connection also formed through the same fasciculus "establishes communication with eye movements and equilibration." Through these communications a reflex arc is formed between eye and head movements in response to vestibular stimulation of the nerve endings in the semicircular canals, through which oscillation of the eyeballs may be induced—nystagmus. A more direct communication through the olivary nucleus explains the auditory reflex of turning the eyes in the direction from which a sound may eminate. The communications between the third and sixth nerve nuclei are well known, as pointed out by Bing and others, in bilateral spasm and paralysis.

The nerve trunk emerges from the lower edge of the pons and above the medulla oblongata, passes upward in close contact to the pons for 15 mm., then beneath the anterior inferior cerebellar artery and penetrates the dura mater centrally to the fifth nerve, and to the inner side of the inferior petrosal sinus. It passes forward through the notch between the posterior clinoid process and the apex of the petrous bone and enters the cavernous sinus to the outer side of the internal carotid artery. The nerve enters the orbit with the oculo-motor nerve, within the annulus of Zinn and the muscle cone, to be distributed to the external rectus muscle on its ocular surface penetrating the muscle in its posterior third.

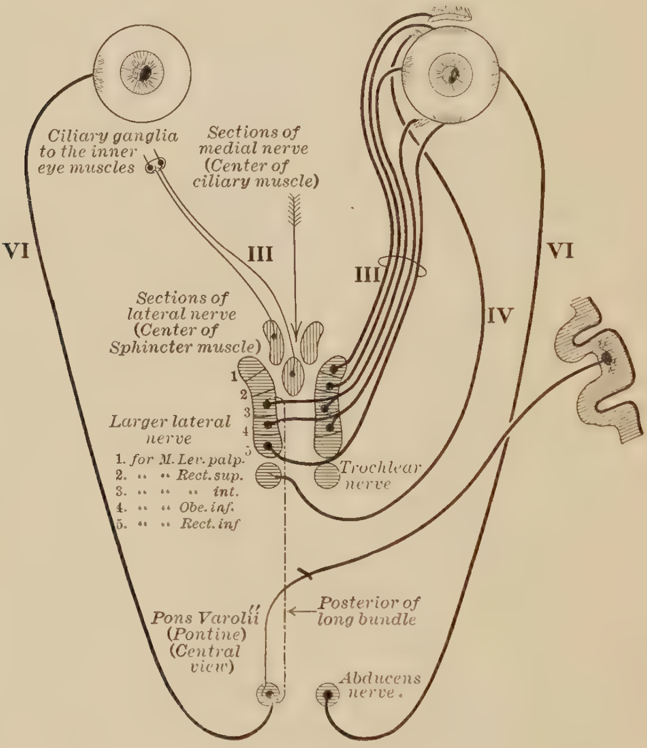
Ciliary ganglia to the inner eye muscles
Sections of medial nerve (Center of ciliary muscle)
VI
III
III
IV
VI
Sections of lateral nerve (Center of Sphincter muscle)
1
2
3
4
5
Larger lateral nerve
1. for M. Lev. palp.
2. " " Rect. sup.
3. " " " int.
4. " " Obe. inf.
5. " " Rect. inf
Trochlear nerve
Pons Varolii (Pontine) (Central view)
Posterior of long bundle
Abducens nerve.

Fig. 22.—*Innervation of the Ocular muscles.* III, Oculomotor nerve; IV, trochlear nerve; VI, abducens nerve. (From Bing's "Regional Diagnosis.")

The exposed course of the nerve within the skull, its compression beneath the anterior inferior cerebellar artery on the pons, and its long course through the dura mater render the nerve peculiarly susceptible to injury in brain tumors and particularly in fractures of the base of the skull and in otogenic pathology. Paralysis of the sixth nerve occurs in a large percentage of fractures of the base in the middle fossa, and in suspected fractures of the base, it is a most important confirmatory symptom. In increased intracranial pressure from brain tumors or inflammatory processes, paralysis of the nerve is also frequently encountered. It is not, however, a symptom of great localizing value in this instance, but rather an indication of increased intracranial pressure. The tumor may be remote from the nerve and at the same time cause sixth-nerve paralysis. Moderate contraction of the pupil on the same side as the paralysis of the sixth nerve, which is occasionally met, is explained by the branches of the sympathetic system which the nerve receives from the carotid plexus in the cavernous sinus. The distribution and the relations of these nerves and their nuclei are most graphically illustrated by Fig. 22, as suggested by Bing.

Variations of the Ocular Nerves.—Variations of the ocular nerves which may have direct bearing on abnormal clinical phenomena occasionally met with, are chiefly the following as outlined by Whitnall.

In the oculo-motor nerve there may be abnormal branches which replace one of the other nerves. Such a branch, entirely or jointly, with the sixth may supply the external rectus, or an extra branch may supply the superior oblique. A branch may anastomose with the sixth. A branch from the inferior oblique may send a double supply to the inferior rectus.

Variations in the fourth or trochlear nerve are recorded. Communications with the orbicularis, supratrochlear, lacrimal, and frontal nerves have been noted.

The sixth nerve has been found totally absent on one side and replaced by a branch of the third. The sixth nerve occasionally enters the orbit outside the annulus of Zinn. A branch to the superior rectus in addition to the supply from the third was recorded by Magath, 1919. For more detailed information on the variation of the ocular nerves, the reader is referred to Whitnall's *Anatomy of the Human Orbit.*

PHYSIOLOGY.

The eye may be regarded as a sphere capable of limited rotations. The center of rotation is 10 mm. in front of the posterior pole and 13.5 mm. behind the anterior pole (Donders).

The geometric or optic axis is an imaginary line which passes through the anterior and posterior poles of the eye including the cardinal points of the dioptric system. $A\ A'$ in Fig. 23. The anterior pole is supposed to be located in the center of the cornea; the posterior pole to the inside of the macula.

The visual line is the line which passes from the point of fixation O to the fovia F in Fig. 23. It necessarily passes through the nodal points *K′ K″*. The visual line and the geometric axis rarely coincide. As a rule, the optic axis is intersected by the visual line and the angle thus formed was described by Donders as the angle alpha (*a*).

The line of fixation is the line which connects the point of fixation *O* and the center of rotation *M* in Fig. 23. This line may or may not coincide with the visual line.

The line of sight is the line which passes through the point of fixation and the pupillary center.

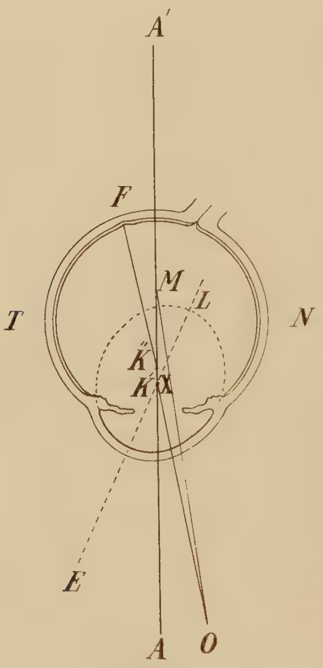

FIG. 23.—*A A′*, optic axis, and in this diagram, also the pupillary axis; *O K′ K″ F*, visual line; *O M*, line of fixation; *E L*, long axis of the corneal ellipse; *M*, center of rotation; *K′ K″*, nodal points of the lens; *F*, macula; *O*, point of fixation; *O X E*, angle alpha; *O M A*, angle gamma; *O K′ A*, angle kappa.

Angles Alpha, Gamma and Kappa.—*Angle Alpha of Donders*, is the angle formed by the intersection of the corneal axis and the visual line. Recently confusion has been added to this question of angles, already complex by reason of numbers, by assigning the term angle alpha to the angle formed by the intersection of the visual line and the greatest axis of the corneal ellipse *O*, *X*, *E* (Fig. 23, Landolt). These definitions of the original alpha of Donders, and the later angle alpha of Landolt, are entirely in harmony, although the phraseology differs. "To this" (the corneal center), says Donders,[1] "in fact, the apex of the ellipsoid of the cornea corresponds."

Angle Gamma (γ).—The angle formed by the lines of fixation $O\ M$, and the optic axis $A\ A'$, or the angle $O\ M\ A$, is known as the angle gamma.

Angle Kappa of Landolt.—The angle formed by the intersection of the visual line and the pupillary axis or the line which passes through the center of the pupil, $O K' A$ in Fig. 23 is known as angle kappa. In the diagram the optic axis and the pupillary line coincide. This angle admits of accurate measurement on the perimeter, has an important bearing upon the measurement of squint and may give the patient the appearance of a divergent or convergent squint when the angle is positive or negative as the case may be. In the interest of simplicity and clearness, it will be the angle considered in this work.

Measuring of Angle Kappa.—The size of angle kappa is best determined clinically on the perimeter. The patient is seated with the eye under examination carefully centered on the fixation point of the perimeter, the other eye being properly occluded. A small electric light of a luminous ophthalmoscope is used as a point upon which the patient fixes, the position of the corneal reflex in the pupillary area is located, the observer being directly behind the light. If the reflex is to the inner side of the pupillary center, the light is moved templeward on the arm of the perimeter until the corneal reflex is found to be in the pupillary center. The observing eye moves with the light while the patient continues to fix in the first position. In this instance the angle kappa is *positive* and the size of the angle is read off on the arm of the perimeter. If the reflex is to the outer edge of the pupil, the light is moved to the nasal side until it reaches the pupillary center and the value of the *negative* angle is determined.

Clinical Value of Angle Kappa.—It is apparent at once that a positive angle kappa will give to a patient the appearance of a divergent squint and a negative kappa will simulate a convergent squint. Furthermore, if a divergent squint is under investigation a positive angle kappa, if present, will tend to increase the angle of squint, and a convergent squint will seem to be less than it really is to the extent of the size of the angle kappa. The converse is true if the angle is negative. In a majority of cases, a positive angle kappa is found in hypermetropia and a negative kappa in myopia. This is the rule but many exceptions are noted.

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[1] On the Anomalies of Accommodation, p. 182.

The value of taking into account angle kappa rather than angle alpha of Donders, lies in the greater ease with which the angle kappa is observed and measured.

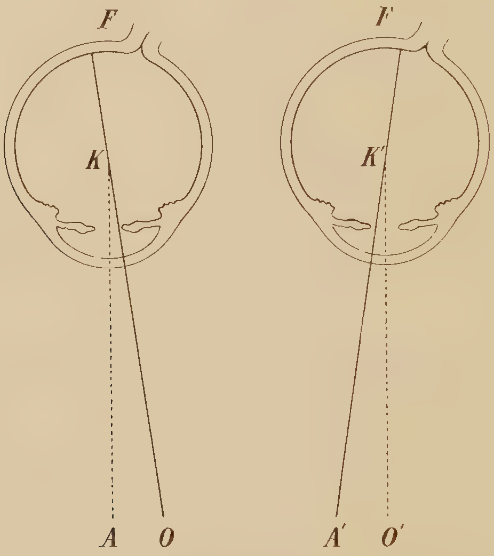

Fig. 24.—Positive and negative angle kappa of right eye.

Ocular Movements.—The eyeball may move in any desired direction. When it revolves around the horizontal axis in an upward deviation, the cornea turning up, the correct term is upward rotation or elevation; if downward, it is downward rotation or depression. When it revolves on its vertical axis, it may rotate externally—temporal rotation; if to the nasal side, the proper terms are internal or nasal rotation. Rotations are possible in all oblique directions, up and out, up and in, down and out, and down and in.

The term *rotation* is used here for these various deviations in order to reserve the term *duction* for a phenomenon which arises in the use of prisms, which will help to keep more clearly before the student's mind the difference between rotations and ductions.[1]

Wheel Motion or Torsion.—In addition to the rotations already discussed, the eyeball, by reason of the action of the oblique muscles, and to a less extent, the action of the superior and inferior recti, may turn on its saggital or antero-posterior axis in a wheel motion which is also called *torsion*. If the upper end of the vertical meridian of the cornea is turned outward or to the temporal side, the condition is extorsion; if turned to the nasal side it is intorsion. It is unfortunate that we do not have a better term to apply to this condition, because of the confusion which arises in the student's mind in differentiation between this wheel motion or true torsion and false torsion to be described shortly. Temporal or nasal wheel motion might be used or better still, right and left wheel motion or rotation might be substituted, but either phrase is cumbersome as compared with intorsion and extorsion. In the use of the term torsion, the reader may infer that by it true wheel motion is meant and the secondary" torsion of Maddox will be called "false torsion." von Helmholtz and his confreres recognized the confusion which arose because of these two conditions—true torsion expressing a movement of the eyeball around the visual axis, and false torsion, a position of the eyeball rather than a movement. In the third volume of von Helmholtz's *Physiological Optics*, many pages are devoted to clarifying the differences, but the uninitiated reader finds "rolling" and "wheel motion" practically the same. The author believes "true torsion" and "false torsion" will be more differentiating and fairly descriptive of the two phenomena.

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[1] This distinction is rarely observed in text-books and may not meet with universal approval. The author believes, however, that if generally adopted the difference in the use of the terms will tend to clearness of expression.

False Torsion.—Simple rotation in any one of the cardinal directions is easily understood. These rotations are accomplished without any torsional movement of the vertical meridian of the cornea. Any oblique rotation, between two cardinal directions is the result of the compounding of two cardinal rotations and each oblique movement is accompanied by a definite degree of false torsion. Donders recognized this fact.

Donders' Law.—Donders, therefore, formulated the following law: "For any determinate position of the line of fixation with respect to the head, thereto corresponds a determinate and invariable angle of (false) torsion, a value independent of the volition of the observer, independent also of the manner in which the line of fixation has been brought into the considered position."

Listing's Law.—A step further is expressed in Listing's law which is as follows: "When the line of fixation passes from its primary to any other position, the angle of (false) torsion of the eye in this second position is the same as if the eye had arrived at this position by turning about a fixed axis perpendicular to the first and second positions of the line of fixation." The law is concisely stated and needs little explanation. It is a provision of Nature, to enable the eye to reach any oblique position by the shortest possible route, saving thereby, time and energy and gaining speed. It does not imply that in passing from primary to secondary position, the eye always follows exactly the same direct course. There may be some variation. It does definitely state, however, that the angle of false torsion always is as though this direct route were followed.

Proof of the Laws of Listing and Donders.—Two experiments will aid the student to grasp quickly the real significance of these two laws.

In a dark room, place a candle on the wall opposite the eye of the observer. The observer looks intently at the light for a few seconds and then rotates the eye up and to the right. The tract of the after-image is readily observed in a graceful curve upward and to the right until the after-image is plainly seen at the end of the line of fixation up and to the right. These lines, whether up and to the right or left or down and to the right or left, are not as a rule perfectly straight, but very slightly curved as shown in the diagram. The lines form the outlines of a sheaf of wheat. They are sufficiently straight, however, to clearly indicate the path followed by the eye in passing from the primary to the secondary oblique position. If instead of a dot of light, a line of light is used, the experiment will in a measure show what the second experiment will show more clearly.

Second Experiment.—A red ribbon, two feet in length, is fastened to a wall vertically, with the center of the ribbon at the height of the observer's eye. The observer should be stationed some distance across the room and should fix attentively on the center of the ribbon for a number of seconds. The eyes are then rotated up and to the right or left. If to the right, the upper end of the after-image will be observed to lean to the right. If the eye is carried to the left, the after-image will incline to the left. When the eye is carried down and to the right, the upper end of the after-image will incline to the left and, if down and to the left, the upper end of the image will lean to the right. If the eye is carried repeatedly to the same secondary point of fixation, the degree of inclination will always be the same (Fig. 26).

The degree of torsion indicated by this experiment is somewhat exaggerated because the wall at the point of secondary fixation where the after-image is seen is not perpendicular to the line of fixation. An apparatus devised by Maddox probably gives one a better idea of false torsion than can be conveyed by words alone. The apparatus consists of a round disk of card-board, from which a rod protrudes at the center, perpendicular to its surface. To the end of the rod is attached a string which suspends a weight (a plumb-line).

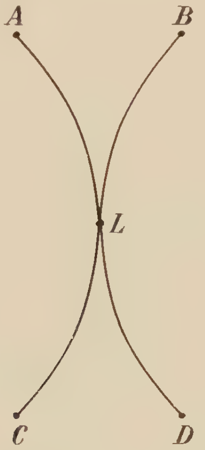

Fig. 25.—The lines form the outline of a sheaf of wheat.

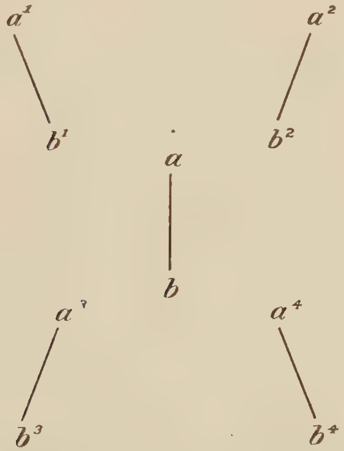

Fig. 26.—Second experiment.

In Fig. 27, No. 1 represents the eye in primary position. Only the end of the extended rod is visible, and the plumb-line cuts the disk at 0 degree below. In No. 2, the eye is turned up and to the right, showing apparently equal and opposite inclinations of the horizontal and vertical meridians *with reference to the fore-and-aft*

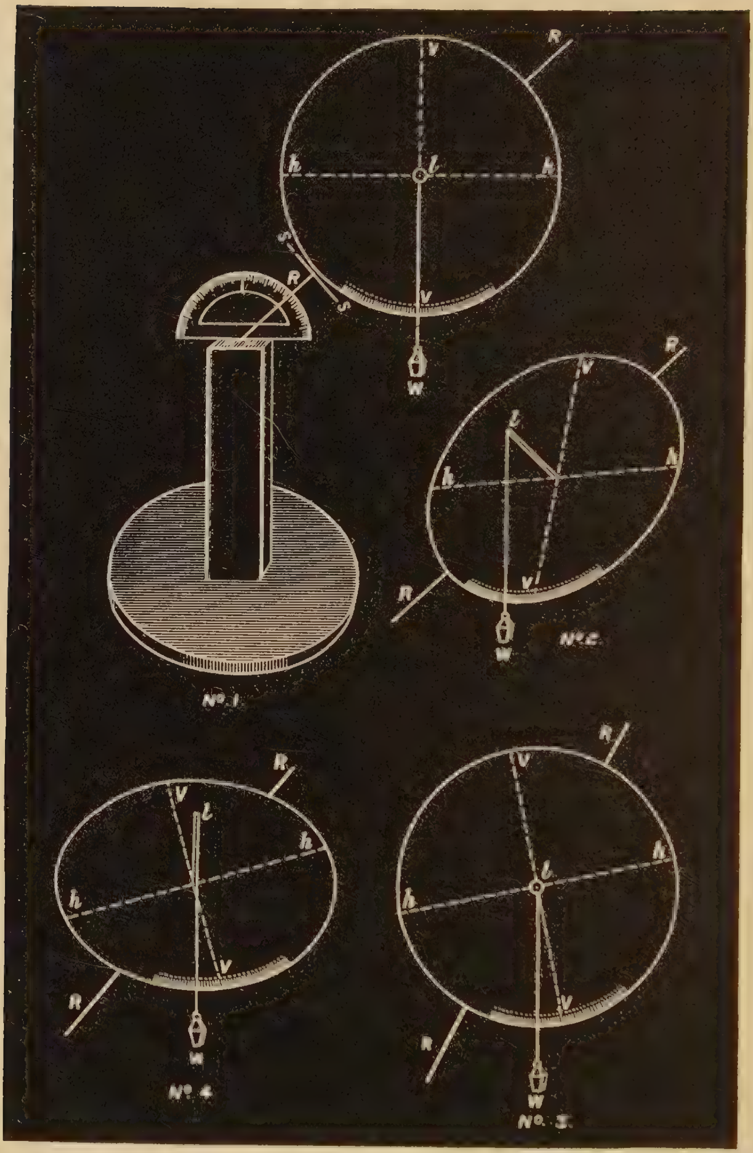

FIG. 27.—The Maddox torsion calculator. No 1, the eye in its primary position. No. 2, the eye looking up and to the right, showing equal and opposite inclinations of the horizontal and vertical meridians with reference to the fore-and-aft axis of the *head*. No. 3, the eye looking as in No. 2 but showing equal similar inclinations of both meridians to the patient's right with reference to the fore-and-aft axis of the *eye*. No. 4, mode of reading same. (From Maddox's "Ocular Muscles.")

axis of the head. The observer's eye has remained in the same position to observe as in No. 1.

To all appearances, the eye as it were is twisted to the left. In order, however, to obtain a proper view of the torsion, the observer's eye should be moved into a position so that the observer's line of vision will coincide with that of the protruding arm, as shown in No. 3. In this position, it will be noted that the vertical meridian has been twisted to the right and the degree of false torsion may be read from the scale at the point where the plumb-line crosses the scale on the rim of the disc below.

The following scale of false torsion from 5 to 40 degrees is taken from von Helmholtz's *Physiological Optics,* vol. 3, p. 49.

Lateral Rotations	Elevation.							
	5°.	10°.	15°.	20°.	25°.	30°.	35°.	40°.
5 degrees .	13′	26′	40′	53′	1° 7′	1° 20′	1° 35′	1° 49′
10 " . .	26′	53′	1° 19′	1° 46′	2° 13′	2° 41′	3° 10′	3° 39′
15 " . .	40′	1° 19′	1° 59′	2° 40′	3° 21′	4° 2′	4° 45′	5° 29′
20 " . .	53′	1° 46′	2° 40′	3° 24′	4° 29′	5° 25′	6° 22′	7° 21′
25 " . .	1° 7′	2° 13′	3° 21′	4° 29′	5° 38′	6° 48′	8° 00′	9° 14′
30 " . .	1° 21′	2° 41′	4° 2′	5° 25′	6° 48′	8° 13′	9° 39′	11° 8′
35 " . .	1° 35′	3° 10′	4° 45′	6° 22′	8° 00′	9° 39′	11° 21′	13° 6′
40 " . .	1° 49′	3° 39′	5° 29′	7° 21′	9° 14′	11° 8′	13° 6′	15° 5′

False torsion is of considerable interest from an experimental standpoint, but inasmuch as it is not of vital importance clinically, the interested reader is referred, for a more detailed study, to Maddox on the *Ocular Muscles,* to von Helmholtz and other books on physiological optics.

Primary and Secondary Positions of the Eyes.—Primary position is the position, best expressed by the phrase "eyes front," a position from which the line of fixation may be moved up or down, right or left, without tilting or torsion of the vertical meridian of the cornea. The phrase "eyes front" when used in this text will indicate primary position as described.

Secondary position may be any other position than primary, which the line of fixation may take.

Action of Individual Ocular Muscles.—Individual ocular muscles never operate singly but in response to innervations in which several or all the muscles share under the coördinating influences of the brain. Only the external and internal recti operating singly are capable of rotating the eye about an axis perpendicular to the visual axis. The individual action of the other muscles, because of their peculiar attachments and the directions of their muscle planes, if operating individually will rotate the globe about an axis far from the perpendicular. Acting, however, under innervational influence, and cerebellar coördination, movements are executed by the combined action of several muscles about an axis which always is perpendicular.

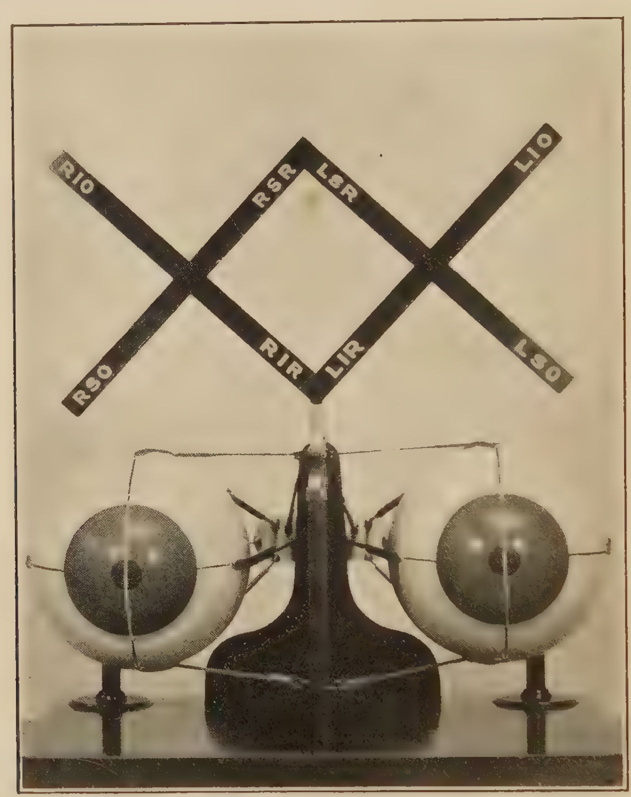

FIG. 28.—Ophthalmotropė, designed by the author and Dr. Edwin B. Miller, with diagram showing the action of the ocular muscles from "eyes front."

Notwithstanding this fact a knowledge of the action of each muscle, from an anatomical standpoint, is essential, in order to fully appreciate the part played by each muscle under innervational influence in executing rotations. Furthermore, it is impossible to clearly conceive of the action of an individual muscle from its mechanical attachments and direction of its muscle plane without regard for the effect upon such action, caused by its relation to the capsule of Tenon and the orbital fascia in general. These have a modifying influence with which one must reckon.

Insertions.—The insertion spiral of Tillaux (Fig. 14), of 5, 6, 7 and 8 mm. in round numbers, indicates the relative distance from the cornea of the internal, inferior, external and superior recti muscles. By this insertion spiral, the internal rectus has the greatest mechanical advantage in purchase, and the superior rectus the least.

Arc of Contact.—The force of each muscle when applied to the globe is a tangential force and is exerted from the point of contact which changes constantly as the eyeball is rotated. In "eyes front," the arc of contact of the internal rectus, for example, extends from the equator to within 5 mm. of the cornea. Maddox has estimated a rotating power of 36 degrees before the attachment of the muscle is ready to leave the contact of the globe. The internal rotating power of the average eye is from 47 to 53 degrees. The difference, therefore, between the estimated rotational power of 36 degrees and that actually encountered clinically, is explained by Maddox by the influence of the attachments of the muscle to the capsule of Tenon, by which the tendon is held in contact to the globe longer than if it were free from all attachments to the capsule.

The arc of contact enters into the question of tenotomies and advancements in the correction of squint. The shortening of the arc of contact in tenotomy of the internal rectus, for example, explains in part the reason for the patient's inability to converge after a tenotomy. It furnishes a valid reason in itself why the internus should not be subjected to a tenotomy in the presence of good central vision in both eyes. Increasing the arc of contact by an advancement operation increases the tangential power of the muscle and is constructive surgery in the correction of squint. The exceptions to this rule will be dealt with in the section on Concomitant Squint.

Primary and Secondary or Subsidiary Functions.—By returning, for a moment, to the anatomy of the ocular muscles, we will recall that the superior and inferior recti have a common origin medially to their insertions. The directions of the muscles from origin to attachments, form an angle with the optic axis of 23 degrees, and according to Landolt, 27 degrees. If the superior oblique be considered as having its origin at the trochlea, from whence its pull must be estimated, the origins of the two obliques are even more medially situated than those of the vertically acting recti. Their planes form an angle, with the optic axis, of 51 degrees. It, therefore, follows that, unlike the external and internal recti, which have but one action (lateral rotation), the vertical recti and the two obliques, in addition to their vertical pull have other functions. The vertical recti, because of their medial origin and forward insertion, must necessarily be internal rotators. The obliques, because of their even greater medial origins and forward pull, and their posterior insertions are external rotators.

Furthermore, because of the medial origin and their medial pull, the superior rectus and superior oblique by reason of their attachment to the upper part of the globe rotate the vertical meridian of the cornea to the nasal side. The inferior rectus and inferior oblique, for the same reasons, by virtue of their inferior attachments to the glove, rotate the upper end of the vertical meridian of the cornea to the temporal side. The former action is called intorsion, the latter extorsion. Manifestly, therefore, these several functions need classification into primary or major functions and secondary or subsidiary.

The external and internal recti have but one action from primary position. The resulting movement is either external or internal rotation. The action of these muscles is totally primary. When the eye is rotated up or down, a subsidiary action of each of these muscles becomes an added function.

The superior rectus has, as its primary function, elevation of the eyeball. Because of its medial origin and forward attachment, as a subsidiary action, it is an internal rotator. Because of its attachment to the upper part of the globe its second subsidiary action is intorsion.

The primary function of the inferior rectus is that of depression or downward rotation of the eyeball. Because of its origin and attachment, like the superior rectus its first subsidiary action is internal rotation; and because of its attachment on the lower surface of the globe its second subsidiary action is extorsion.

Insofar as we have analyzed the functions of the four recti it is evident that the cardinal rotations up and down, out and in, are dominated in each instance by a single muscle. While other muscles share in these rotations under innervational influence, from an anatomical standpoint, the superior rectus, for example, is better fitted to elevate the eye than any other muscle. The same is true of each of the other recti in their particular fields.

Because of the advantageous origins and attachments of the obliques, they are better adapted to execute wheel motion or torsion of the globe than any other muscle. To the obliques, therefore torsion is assigned as a dominant function, with elevation or depression and external rotation as subsidiary actions. In designating torsion as a primary function, we are not unmindful of the fact that these muscles have an elevating and depressing power, second only to the vertical recti. That their vertical pull is second to that of the recti, however, is undisputed even by those who claim that the vertical pull should be considered as a primary action. In assigning torsion to the obliques as a primary function and elevation and depression as secondary, the author finds Maddox is entirely in accord. The reader may recall that the obliques are attached to the posterior and outer surfaces of the globe and that the muscle planes form an angle of 51 degrees with the median plane of the head. Their advantageous attachments, therefore, make them superior as torsion muscles to the recti which form an angle of 23 degrees with the median plane. This oblique angle of attachment also increases their power of external rotation. These two functions, therefore, are at the expense of their vertical pull. Furthermore, when we consider the number of rotations which the eyes execute outside of the median horizontal plane, the function of torsion assumes the proportion of a major role. For these reasons the author has committed himself to the policy of attributing to the obliques the major role of torsion.

TABLE OF PRIMARY AND SUBSIDIARY ACTIONS OF INDIVIDUAL OCULAR MUSCLES.

Muscle.	Primary action.	Subsidiary action.
Internal rectus	Internal rotation	None.
External rectus	External rotation	None.
Superior rectus	Elevation	Intorsion. Internal rotation.
Inferior rectus	Depression	Internal rotation. Extorsion.
Superior oblique	Intorsion	Depression. External rotation.
Inferior oblique	Extorsion	Elevation. External rotation.

Relation of Primary and Subsidiary Functions.—Subsidiary functions are always at the expense of the primary function and *vice versa*. This is easily shown, for example, in the action of the superior rectus. In primary position, the superior rectus elevates the eye primarily and secondarily rotates it to the nasal side and intorts the cornea. When, however, the eyeball is turned out, the primary or elevating power is increased and the subsidiary actions of internal rotation and intorsion are diminished. When the outward rotation reaches 23 degrees, and the muscle plane and optic axis coincide, the subsidiary actions of the muscle totally disappear and the kinetic energy of the muscular contraction is spent solely in elevation. On the contrary, if instead of external rotation, the eye is rotated from primary position to the nasal side or in internal rotation, the subsidiary functions of the muscle increase at the expense of elevation or the primary function.

The same rule applies to the inferior rectus. For these muscles, therefore, the following rule is established. In external rotation the vertical power increases. Conversely, in internal rotation torsion and internal rotation increase and the vertical pull diminishes.

The general laws governing the relation of primary and secondary functions of the recti apply also to the obliques with modifications. The torsional or primary function as well as the power of external rotation increase as the eye is rotated to the temporal side, and theoretically, reach their maximum when the eyeball has rotated outward through an arc of 39 degrees at which point the vertical pull becomes *nil*. When, on the other hand, the eye is rotated to the nasal side, the elevating or depressing power increases and should reach its maximum when the eye is rotated through an arc of 51 degrees, while at this point torsion and external rotating power should entirely cease.

It will be noted, therefore, that the vertical pull of the recti muscles increases in external rotation and that of the obliques in internal rotation. This is one of the important facts to be remembered as it has a valuable clinical application in the differential diagnosis of palsy of the vertical muscles. It will, also, help the student to understand the significance of and the reasons for grouping the vertically acting muscles into right- and left-hand elevators and depressors.

In Fig. 29 an attempt is made to convey to the reader a diagram of the action of the individual ocular muscles from primary position. This figure will be referred to throughout the text and, if properly fixed in the reader's mind, will simplify many of the otherwise complex problems with which one is brought face to face in a study of ocular muscle palsies. The anatomical method of recording is followed. Right and left refer to the patient's right and left. It is the logical manner in which a patient is examined, the examiner standing in front of and facing the patient.

Synergistic Action of Muscles. The eye is never moved by the action of a single muscle. Even so simple a rotation as that of turning the eye to the right or left in a horizontal plane probably involves the combined action of three muscles. These aids to the action of a single muscle are called synergists. The synergists to the internal rectus, for example, in internal rotation are the superior and inferior recti. Aids to the external rectus in external rotation are the superior and inferior obliques.

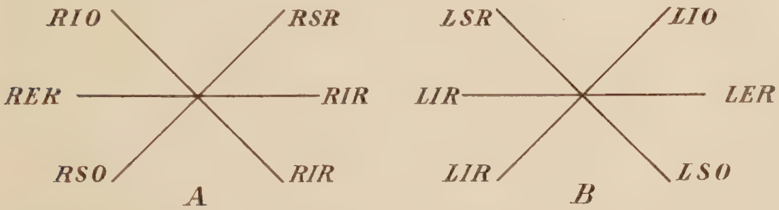

FIG. 29.—Action of the individual ocular muscles from primary position.

The synergistic action of the ocular muscles can be readily grasped by referring to Fig. 29. In this figure, the actions are graphically recorded.

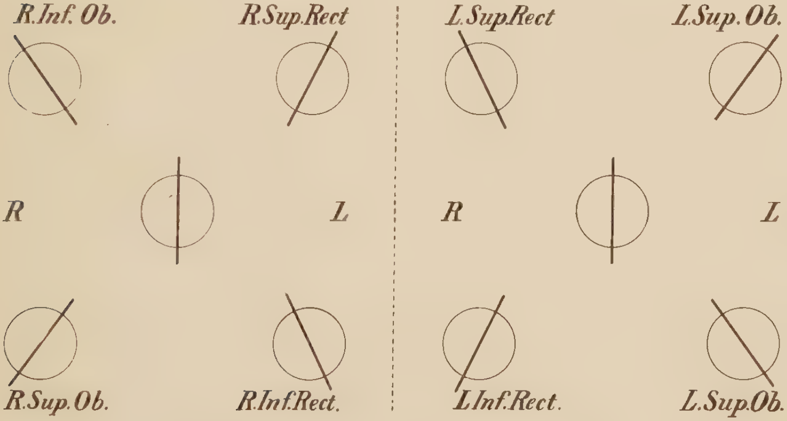

FIG. 30.—Normal action of individual muscles from primary position or from "eyes front."

TABLE OF SYNERGISTS.

Muscle.	Synergist.
Internal rectus	Superior rectus. Inferior rectus.
External rectus	Superior oblique. Inferior oblique.
Superior rectus	Inferior oblique. Internal rectus.
Inferior rectus	Superior oblique. Internal rectus.
Superior oblique	Inferior rectus. External rectus.
Inferior oblique	Superior rectus. External rectus.

In addition to the table of synergists given above, further synergistic action is possible. For example, in a straight upward pull the superior rectus is naturally the dominating muscle and the inferior oblique the synergist. If, however, the rotation is up and out the inferior oblique, at first the major muscle, assisted by the superior rectus and external rectus, gives way at a given point in the rotation as a major muscle to the superior rectus, and assumes the role of a synergist. The reason for this transfer of the major function lies in the fact that the superior rectus acts best as an elevator when the eye is in a position of external rotation. The inferior oblique, on the other hand, diminishes in power as an elevator in this position. It may be less confusing to the student, in analyzing the up and out rotation, to regard the superior rectus as the major muscle and inferior oblique and external rectus as synergists or to regard the external rectus, superior rectus and inferior oblique as synergistic muscles, in this rotation, without regard to the major or minor role assumed by each muscle. In fact, in the illustration just given it would be seemingly impossible to assign a major role to any one of the three muscles, if it were not recalled that in this up and out rotation the total energy of the superior rectus is spent in upward rotation, while that of the oblique is divided between an upward and an external rotation. The same may be said of the external rectus. This is an external rotator, but when the eyeball is rotated upward in a secondary position some of its energy is lost for external rotation and is spent in a vertical direction.

Wheel motion or torsion is also brought about, not by a single muscle but by the synergistic action of, at least, two and probably more muscles. In this action the obliques assume the major role. The vertical recti, however, are also torsion muscles, but secondary in efficiency in primary positions. In certain secondary positions their relative intorting or extorting power may be reversed. When the eyes are turned nasalward the torsional action of the recti increases and that of the obliques decreases, and the converse is true when the eye is in a position of external rotation.

The possibilities in variation in the synergistic action of the ocular muscles is very great, as one can comprehend from the many secondary positions assumed by the eye. The thought, however, which I wish to leave with the reader, in discussing synergistic action, is the fact that an ocular muscle never acts alone, even in the most simplified movement, but is assisted by those muscles which have similar actions and these muscles are known as synergists. This applies to primary and equally so to subsidiary actions.

Antagonists.—Every ocular rotation, as previously pointed out, is the result of a central innervation and such rotation is inseparably associated with a similar rotation in the fellow eye. Innervations are bilateral in their action. In the discussion of synergists, however, binocular action is totally ignored, as it will be in the discussion of antagonists.

If there are muscles which assist because of similar actions, there are others whose actions are diametrically opposite, either in primary or subsidiary functions or both. These opposed movements may be in the nature of a check or a stabilizing character, or they may be direct antagonists under certain conditions. The external rectus, for example, may check the overaction of the internal rectus, or the inferior oblique may neutralize the torsional action of the superior oblique. On the other hand, the external rectus as a direct antagonist of the internus will produce external rotation of the eyeball if the internus is paralyzed. In either sense the action is that of an antagonist. The vertically acting muscle may, at the same time, be synergistic and antagonistic in action. The superior rectus is the direct antagonist of the inferior in vertical pull and in torsional action. In internal rotating power these muscles are synergists both being internal rotators. The inferior oblique is a synergist to the superior rectus in elevating action but an antagonist of the same muscle in torsional action and in lateral rotation. The two obliques are antagonists in torsion and in vertical rotation, but synergists in external rotation.

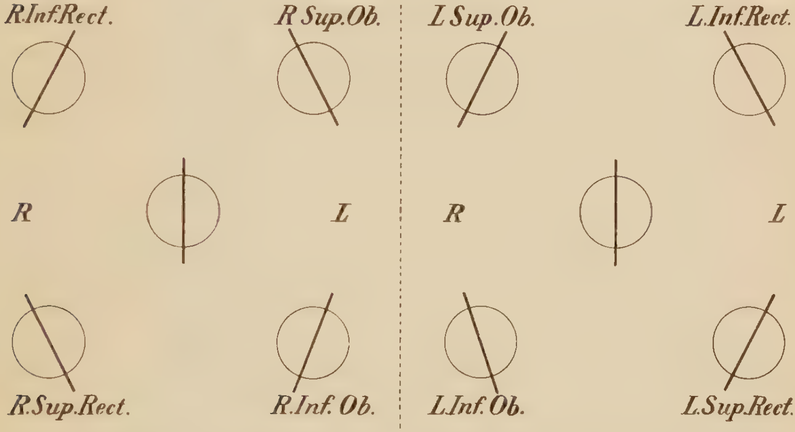

Fig. 31.—Primary deviation of the eye in individual muscle paralysis as the result of the action of antagonists.

The following table of direct antagonists may be supplemented by the reader for other positions than primary. The combinations possible are many and interesting.

TABLE OF DIRECT ANTAGONISTS.

Muscle.	Antagonists.
Internal rectus	External rectus. Superior oblique. Inferior oblique.
External rectus	Internal rectus. Superior rectus. Inferior rectus.
Superior rectus	Inferior rectus. Superior oblique.
Inferior rectus	Superior rectus. Inferior oblique.
Superior oblique	Inferior oblique. Superior rectus.
Inferior oblique	Superior oblique. Inferior rectus.

Conjugate of Yoke Muscles.—Thus far the discussion of muscle action has been limited to the muscles of a single eye. This was necessary in order to grasp the full possibilities in the function of each muscle. When the action of each muscle is reduced to a diagram, such as that shown in Fig. 29, and the diagrams of the two eyes are compared, one is impressed at once with the fact that in each eye there is a muscle whose actions are similar to a certain muscle in the other. This applies not only to primary but also to subsidiary functions. In fact, if the figure is carefully examined it will be noted that all the muscles are arranged in pairs. When the action of individual muscles is carefully analyzed it becomes even more apparent that certain corresponding muscles in the two eyes have actions which are identically the same; the supposition becomes manifest that they are intended to act together, even though no mention has been made of a nervous impulse which might cause these corresponding muscles to respond similarly to the same impulse. To these muscles so similar in action the name *yoke* or *conjugate* muscles has been most appropriately given.

The least complicated of these muscles in action are the internal and external recti. The right externus and the left internus, the right internus and the left externus are similar in action in that the former group are right-hand rotators and the latter group are left-hand rotators. In the vertical recti and the obliques the similarity is more striking.

The right superior rectus elevators, intorts and rotates the right eye to the left. The left inferior oblique elevates, extorts and rotates the left eye to the left. In other words, these muscles are elevators, left-hand rotators and in wheel motion cause the vertical meridians of the corneæ to lean to the left. They are the actions of the muscles from primary position. In secondary positions the similarity continues. When the eyes are turned to the right from primary position the elevating action of the right superior rectus increases and intorsion and internal rotating power diminish until right-hand rotation reaches a point 23 degrees from primary position, or until the muscle plane coincides with the optic axis, when torsion and the internal rotating power cease and elevation is *ad maximum.* Because of the elevation of the eye *ad maximum* in right-hand rotation, Duane called the right superior rectus a right-hand elevator.

The left inferior oblique has a similar action on the left eye. When the left eye is rotated to the right, the elevating action increases and left-hand torsion or extorsion of the vertical meridian and left-hand rotating power of the inferior oblique diminish. To this muscle Duane gave the name of *right-hand elevator* of the left eye. The right superior rectus and the left inferior oblique are yoke muscles. They are right-hand elevators.

The fact that the muscle plane of the right superior rectus forms an angle of 23 degrees with the optic axis and the muscle plane of the left inferior oblique forms an angle of 51 degrees with the optic axis of the left eye does not invalidate the similarity of action. This difference in mechanical elevating power because of the difference in the angle of attachment is equalized by the action of the synergists and antagonists in each eye, in executing binocular movements. The end-result in elevation of each eye is the same, although the mechanical means by which elevation is obtained differs in the two eyes. The bilateral movement is the result of a central innervation in the execution of which probably all of the ocular muscles share, but in which the right superior rectus and the left inferior oblique are the dominating muscles.

The left-hand elevators are the left superior rectus and the right inferior oblique. These muscles are elevators, rotate the corneal meridians to the right and draw the eyes to the right from primary position. When the eyes are turned to the left their elevating power increases at the expense of right-hand torsional action and right-hand rotation.

A similar analysis of the depressor muscles separates this group into right-hand depressors, the right inferior rectus and left superior oblique and left-hand depressors, the left inferior rectus and right superior oblique.

The right inferior rectus and left superior oblique from eyes in primary position depress the corneæ, rotate the upper meridians to the right and, in addition, turn the eyes to the left. When the eyes are turned to the right depression increases at the expense of right-

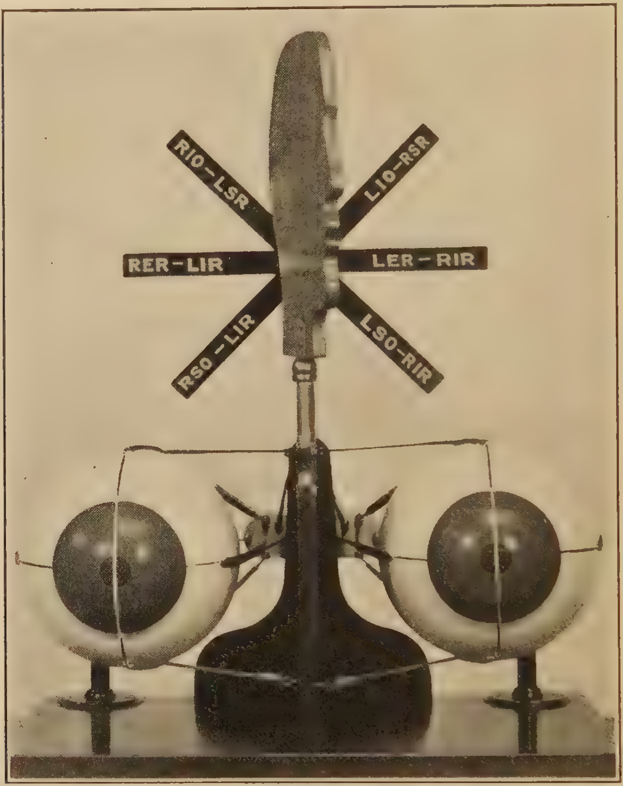

FIG. 32.—Ophthalmotrope, with a diagrammatic grouping of conjugate muscles.

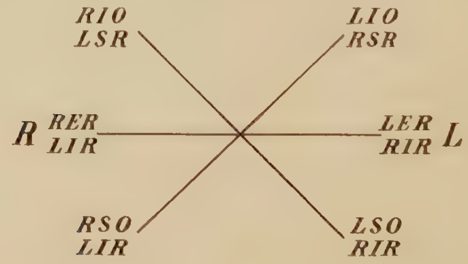

FIG. 33.—Yoke or conjugate muscles.

hand torsion and left-hand rotation. They, therefore, are right-hand depressors.

From primary position, the left inferior rectus and right superior oblique depress the corneæ, rotate the corneal meridians to the

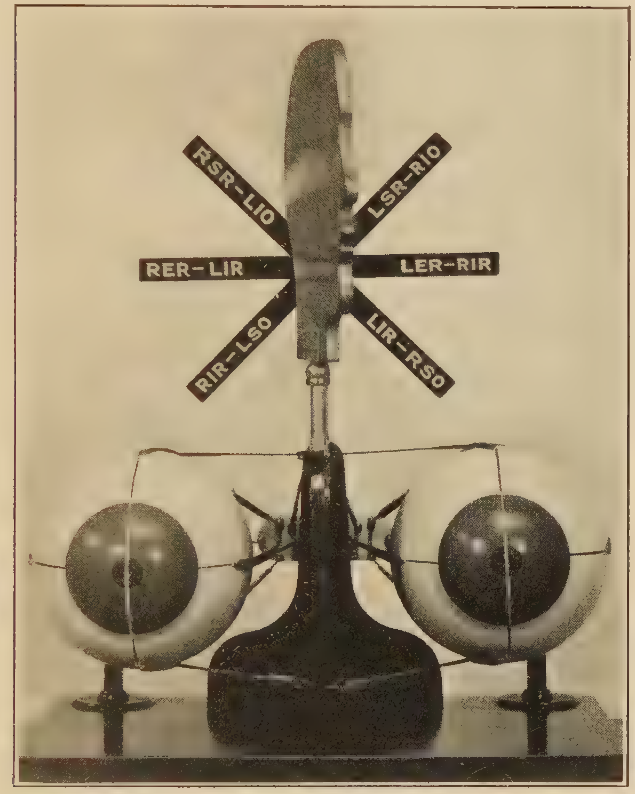

Fig. 34.—Ophthalmotrope with muscles grouped as right- and left-hand elevators and depressors, and right- and left-hand rotators.

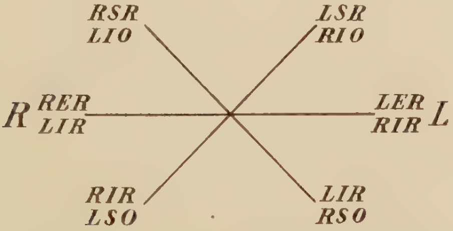

Fig. 35.—Yoke muscles grouped as right- and left-hand elevators and depressors.

left and rotate the eyes somewhat to the right. When the eyes are secondarily turned to the left, inferior rotation increases while left-hand torsion and right-hand version diminish. They are left-hand depressors.

The conjugate grouping of the muscles of the two eyes is most readily grasped by superimposing *A* and *B* of Fig. 29, with the following results:

In Fig. 33 the grouping of the muscles of the two eyes is simply brought about by superimposing *A* and *B* in Fig. 29. Inasmuch as reference has been made to Duane's classification into right- and left-hand elevators and depressors, it will aid the student to group these muscles diagrammatically. In Fig. 34 the muscles are arranged as right- and left-hand elevators and depressors, and into right- and left-hand rotators. It will be noted that the right superior rectus and the left inferior oblique are the right-hand elevators of the two eyes. In the diagram these are directed up and right. Each of the other groups are similarly drawn (Fig. 35).

Innervations.—From what has been said of the action of individual muscles, of synergists, antagonists and of conjugate muscles, it is evident that the combined actions of the ocular muscles, in executing a movement, require a center in which the innervation originates and a coördinating center. As one muscle never acts singly, so in ordinary vision one eye never acts alone but both eyes move as one eye. In certain pathological conditions but one eye may respond to an innervational impulse, the failure of the other to move being due to the pathological condition present, or to an unusual volitional act on the part of the individual It is possible for certain individuals to place one eye in a secondary position, as in convergent squint. This is unusual and abnormal.[1]

Conjugate Movements.—Of the many associated movements of the eyes which have been thought to be sufficiently definite to indicate the presence of specific centers, five at least are accepted by all as definitely determined. They are the conjugate movements of the eyes to the right and left, up and down, and in convergence. Movements to the right and left originate contralaterally. The centers for movements of the eyes to the left are on the right side of the brain and centers for movements to the right are on the left side of the brain. A destructive lesion in either center, or along the course of the paths leading from these centers to the nuclei of the third, fourth and sixth nerves in the floor of the fourth ventricle,

[1] A case recently reported by Holloway and Spiller was able, at will, to cause either eye to diverge.

will result in conjugate deviation toward the side of the lesion. If the lesion is on the right side, movement to the left will be impossible and the eyes will deviate to the right. If the lesion is on the left side, deviation to the right will be impossible.

Centers for binocular elevation and depression are undisputed as well as that of convergence.

Divergence is often spoken of as negative convergence. There are recorded in the literature, however, many cases of paralysis of divergence. This condition is so distinctly definite with a characteristic symptomatology, and, although a center for the same has not been located, it is probable that a center for this movement exists and that this conjugate movement should be regarded as the sixth.

Maddox is inclined to believe that bilateral right- and left-hand torsion should be recognized as definite conjugate movements. He admits, however, that these movements have never been observed in nystagmus. Similarly there may be centers which may cause the vertical meridian of the corneæ to incline toward or to diverge from each other. Insofar, however, as is definitely known, but five conjugate movements are recognized with a strong possibility of a sixth, namely, divergence.

Innervation Centers.—Centers for voluntary lateral conjugate movements are located, in all probability, in the upper part of the mid-frontal convolution of the cerebrum.

Involuntary centers for these movements are probably located in the cuneiform body along the calcarine fissure. Visual centers in the right cuneus receive impressions from corresponding points on the right halves of each retina, *i. e.*, from the left field and *vice versa.* Experimentally, Schafer and Monk found that stimulation of the forepart of the visual centers in the calcarine fissure caused depression of the eyes and stimulation of the posterior part of the fissure produced elevation. They even found a point, stimulation of which caused neither depression nor elevation but in some instances convergence. Much of our knowledge is purely of an experimental character. We reason from analogy by experiments on lower animals and draw conclusions for the human race. They may and may not be correct. However, it is generally conceded that the involuntary conjugate centers are located in the occipital lobes.

Centers for Convergence and Up-and-Down Movements.—Conjugate centers for voluntary movements to the right and left have been definitely located in the upper end of the mid-frontal convolutions. Centers on the right direct conjugate movement to the left, and centers on the left control movements to the right. Electrical stimulation of these centers produces conjugate ocular movements as indicated above. Furthermore, Festa[1] elicited similar conjugate movements by stimulating the "*area pre-occipitalis*" in the region of the angular gyrus. It is probable, however, that these latter areas transmit intercommunicating fibers from the cortical centers located in the mid-frontal convolutions.

No other conjugate centers have thus far been located. The most searching investigation, either experimentally or pathologically, has thus far failed to uncover such centers. Paralysis of divergence, convergence and up-and-down movements are often found in disease along the aqueduct of Sylvius and in the region of the anterior corpora quadrigemina. In these areas the transmitting fibers are easily caught in the posterior longitudinal bundles. However, there should be and probably are cortical centers for these movements, and the most that can be said of them is "They have thus far not been uncovered."

The *coördinating center* for ocular movements is in the cerebellum. This body serves as a coördinating center for all synergetic movements of the body. Ocular centers, according to Stewart, are located in the anterior end of the vermis close to the median line. Cerebellar lesions involving this center are accompanied by nystagmus, sometimes by conjugate deviation of the face and eyes to the side of the lesion, because of the loss of cerebellar control of ocular movements through Deuters' nucleus. It is patent, therefore, that conjugate innervations are under the control really of conjugate centers (the volitional centers in the mid-frontal convolution), the involuntary centers in the occipital lobes and the coördinating centers in the cerebellum. Perfectly coördinated conjugate movements, therefore, are dependent not only upon intact conjugate centers but also upon uninterrupted intercommunicating fibers and unobstructed pathways from these centers to the nuclei of the ocular-motor nerves in the floor of the fourth ventricle. Disturbance in any part of this complex conjugate circuit results in some form of conjugate disturbance—palsy or spasm. The only exception to be noted is a lesion in the nucleus of the sixth nerve, reference to which will be made in the section on Conjugate Paralysis. Lesions in the nuclei themselves, with the exception noted, or below the nuclei result in single or mutiple palsies of individual muscles.

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[1] Deutsch. Ztschr. f. Nervenheilk., 1926, **94**, 45.

Binocular Vision.—Binocular rotations are intended to bring about and facilitate binocular vision. It is the only reason for the existence of conjugate movements. Before studying the essential phases of binocular vision, it will be of interest to pause for a moment in a brief study of the position of the eyes in sleep and during wakeful hours when they are ready to respond to innervational influence.

Position of Eyes During Sleep.—During sleep or unconsciousness from other causes all innervational influences are absent or in abeyance unless pathological control of some part of the motor pathway changes the natural order of things. The eyes, therefore, may be said to assume a position of *anatomical rest.* This position usually is up and out.

Functional Rest.—During conscious moments, when the eyes are not responding to some innervational influence and are not fixed in attention, they may be said to be in functional rest because, although ready at a moment's notice to "stand attention," so to speak, the position which they assume requires no effort. The relation of "anatomical rest" to "functional rest" is not unlike the relation of "at ease" in military parlance to that of "at rest." In the first instance the line is broken, but the individual members of a company are within reach of the order "fall in." "At rest," on the other hand, the individuals are ready to respond to the order "attention," from which movements are executed.

Much interest attaches to the state of the muscles during the period of functional rest. Are they in a state of complete relaxation or is there a certain amount of tonicity present at all times? It is probable that the latter theory prevails. In any innervational act certain muscles are called into action while others, in order to assist properly, must be in a state of relaxation. It is probable, therefore, that in order to carry out an innervation a general condition of equal tonus is present in all the muscles. This, as Hansell and Reber have pointed out, will allow the proper amount of relaxation of antagonistic muscles to keep pace with the contraction of the other muscles.

Fixation.—During conscious moments the eyes may wander from object to object without special attention to any single object or they may suddenly fix intently upon a definite object in order to obtain better knowledge of the same. The object which engages the eyes in fixation is called "the point of fixation." The eyes are said to "fix." It is believed that a reflex fixation center is located in the region of the corpora quadrigeminal bodies. The object which engages our attention may be too large to "fix" and we, therefore, go from point to point on the object, each new point becoming the point of fixation.

The *line of fixation* is an imaginary line which connects the point of fixation with the center of rotation of the eyeball.

Direct and Indirect Vision. In fixation the most sensitive part of the macula, the fovea centralis, is used to focus upon the "point of fixation." For some distance beyond the fovea the macula is highly developed and has good differentiating ability, but in the fovea centralis alone does visual acuity reach its highest degree of development. According to Tscherning,[1] "The breadth of the anatomical fovea is 0.2 to 0.4 mm., or, viewed from the posterior nodal point, which is 16 mm. from the retina, it subtends an angle of from 45 minutes to 1 degree and 30 minutes." The point of fixation, however, has a much smaller breadth. "Central fixation" means fixation of an object on the most sensitive part of the fovea. In defining direct vision, therefore, one is apt to regard macular vision as synonymous. Strictly speaking, central vision is concerned with the most sensitive part of the macula, namely the fovea.

Indirect Vision.—Macular sensitivity recedes rapidly from the fovea to the periphery and beyond the macula the rods and cones become fewer and retinal sensitivity gradually diminishes to the most peripheral boundaries of the retina. Although less sensitive than the fovea, the extra-foveal retina has a special function of recognizing moving objects. One of the special functions of the peripheral retina engaged in indirect vision is to call attention to objects outside of the line of fixation, especially moving objects, in order that we may "fix" them with direct vision and determine their real character.

We are concerned to a considerable extent with indirect vision in a study of ocular muscles, as, for example, in orienting ourselves in relation to objects not in line of direct vision, in physiological diplopia, etc., but special attention centers on *direct* or foveal vision.

Field of Fixation.—The field of fixation may be defined as the base of a cone, the apex of which is the center of rotation of the eyeball, as the eye is rotated to its extreme limits in all directions, foveal vision at all times marking or defining the base of the cone. Indirect vision does not enter into the defining of the field of fixation. It is concerned with central vision only. It is like the visual field, however, for form and colors, in that the nose does not admit of a proper recording of the rotation down and to the nasal side and to some extent upward and to the nasal side. In binocular fixation, however, as in measuring the extent of binocular visual fields, one eye continues to function when the fellow eye is no longer able to fix because of the obstruction offered by the nose. The field of fixation is taken for each eye separately.

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[1] Physiological Optics.

Methods of Measuring the Field of Fixation.—Two methods may be employed. The first, and the one usually adopted, is that by means of the perimeter and the second, now rarely practised, is by Steven's tropometer. The perimetric method is satisfactory, accurate and easily executed. Great care, however, must be observed in noting that the patient's head is at all times in the same position and that the patient shall fully understand the object sought in the test. To this end he should be thoroughly instructed to the minutest detail. One eye is occluded by an eye-pad or bandage. He is seated before the perimeter with the eye under investigation carefully centered over the eye-piece supported in front of the chin-rest, and fixed on the center of the perimeter. Illumination should be good as for reading purposes, although standard illumination, such as is employed in perimetry, is unnecessary. The best form of test object consists of a few printed letters on cardboard, the letters being sufficiently large for even a presbyope to be able to read comfortably at 33 cm. Instead of letters, figures may be employed or four dots or two lines properly spaced, so that they may be distinctly seen, may be found satisfactory. The test object is slowly moved along the arm of the perimeter up or down, and the patient is instructed to follow the test object with his eye to the extreme point of rotation, the letters being clearly seen. If blurring occurs the test object has been moved too far for foveal vision, and it should be moved back a trifle until the letters again become distinct. This point, as read off from the arm of the perimeter, marks the extreme rotating power of foveal vision in that direction, and the result may be recorded as shown in the chart (Fig. 36), or it may be recorded on a perimetric chart, as shown in Fig. 37. The test object is now brought back to the center of the perimeter and the opposite field of rotation is taken in a similar manner and the results are again recorded. The technic of lateral and oblique rotations differs slightly from the up-and-down rotations. On taking right-hand rotations the patient should be instructed to center his attention on the last letter on the card in order to make accurate measurements, and in rotating the object to the left the first letter should be fixed. For purposes of uniformity, the student is urged to use the anatomical method of observation rather than the projected field of rotation. This method will be observed throughout this volume. Note carefully Fig. 36 with reference to the patient's right and left.

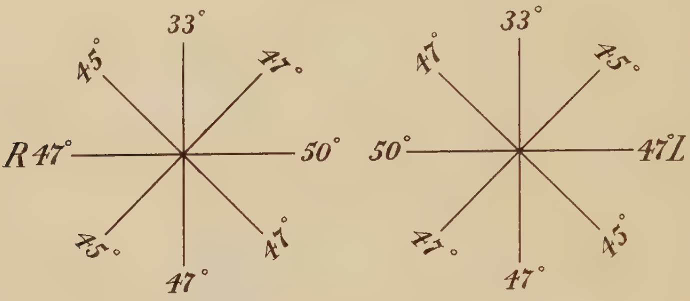

Fig. 36.—Author's method of recording fields of rotation.

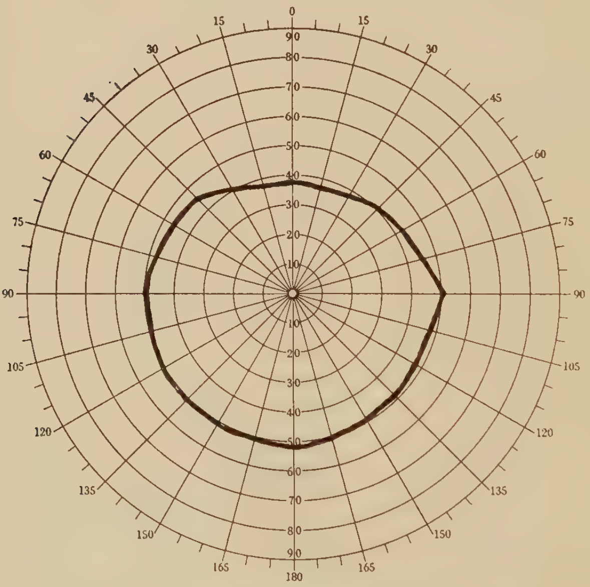

Fig. 37.—Method of recording field of rotation on a perimetric chart.

The method devised by Stevens by means of his tropometer, on the whole, is not nearly so satisfactory, and it entails the purchase of another instrument at a considerable expense. In the author's experience, the tropometer offers greater latitude for variations than the perimeter, and is consequently more liable to error. For

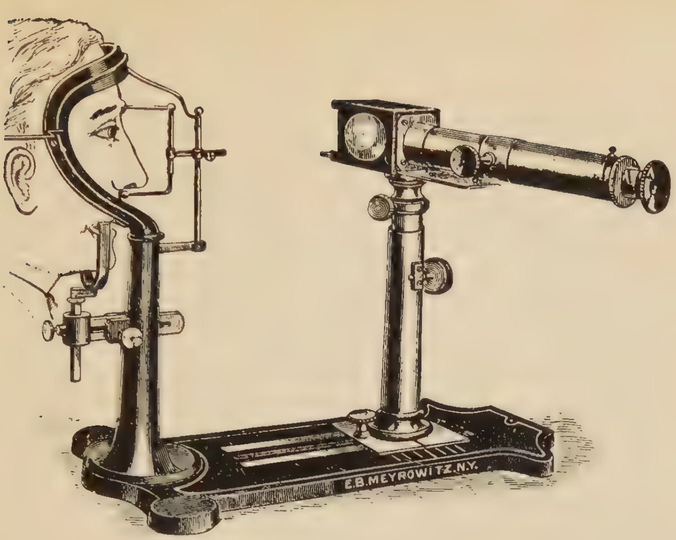
E.B.MEYROWITZ.N.Y.

FIG. 38.—Steven's tropometer consists essentially of a telescope in which the inverted image of the examined eye is found at the eye-piece, where its movements can be accurately observed. A graduated scale in the eye-piece permits every movement of rotation, in any direction, to be exactly measured. The patient's head is fixed immovably in a chin-rest.

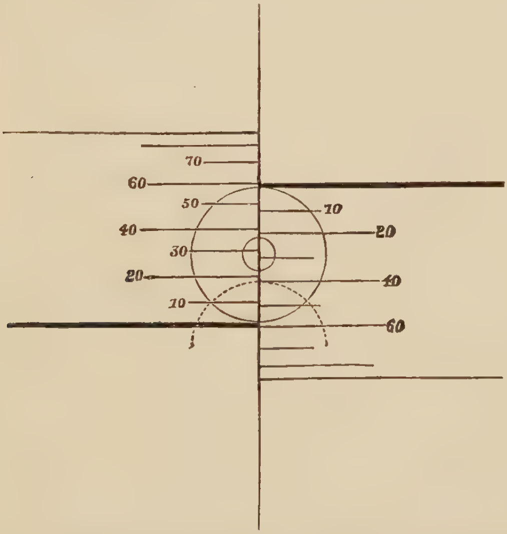

FIG. 39.—Scale of Steven's tropometer. The long line between and at right angles to the shorter lines divides two similarly graduated scales running in different directions; the larger circle represents the outer border of the cornea, the edges of which are in contact with the two strong lines; the interval between each pair of short lines of the scale is 10 degrees of arc, commencing at the strong line in each case. If now the head of the person examined is held firmly in the primary position and the eye caused to rotate strongly in a given direction, the arc through which the border of the cornea passes may be accurately read upon the scale. In the figure, 39, the curved dotted line, represents a new position of the border of the cornea.

Suppose that the person examined has been directed to look strongly upward, then the cornea has moved down and scale and reaches the point in this example of 40 degrees, that being the measure of rotation. By means of the small lever the scale can be placed horizontally, vertically, or obliquely, and by means of the two graduations measurements in opposite directions can be made.

details as to its operation, the interested reader is referred to Stevens' book, *Motor Apparatus of the Eye.* Other methods for recording fixation fields have been devised from time to time. The Greens, from San Francisco, presented an instrument, designed by them for this purpose, at the meeting of the Ophthalmological Section of the American Medical Association in 1926. Fairly accurate results may be obtained by this instrument, but it was designed as a clinical instrument to indicate the paretic muscle rather than to accurately determine the extent of the paresis. It possesses possibilities, nevertheless, of being shaped into an instrument of much precision.

Variations in Rotations.—The rotations in the various directions differ in individuals normally. The average rotations in the cardinal directions are as follow: Upward, 33; downward, 47; inward, 50; outward, 47.

According to Stevens, although the lateral breadth of rotations is greater than the vertical, there is greater normal variation in the vertical than in the lateral. The figures as given above may be considered as the normal minimum measurements. Any marked digression from these figures on the proximal side should be regarded as defective rotations.

Number of Meridians to be Studied.—In the average case a study of the four cardinal directions will be sufficient. In suspected weakness or overaction of the obliques and the vertical recti, the oblique meridians bisecting the cardinal rotations should also be studied. This is also necessary in differentiating between defective rotations due to an ocular palsy and those due to squint of the concomitant variety. Reference to this difference will be made in the section on Paralytic Squint.

Fundamental Laws.—*Laws Governing Projection and Direction.*—In order to fully understand the complex visual act, certain functions of the percipient nerve elements in the retina and the optic nerve itself require careful analysis. It is a function of the sensory nerves of the body to refer their sensations, not to the immediate nerve itself, if stimulated, but to its peripheral distribution or to the member to which it gives sensation. The pain from coxalgia is not felt, as a rule, in the hip, but it is referred to the inside of the corresponding knee. Of a more complex character is the pain which is sometimes experienced in pneumonia, felt not in the lung, but reflexly referred to the homolateral iliac region. These instances are cited in order to show that the projection of the image recognized by the retina is not a peculiar phenomenon, but a condition which is common in sensory nerves. The image which is impressed upon the rods and cones of the retina is not recognized by the gray matter of the brain as a sensation on the retina, but a phenomenon which is projected into space. The direction which the image will take in space is controlled by the law of direction.

In Fig. 40 the points in space, *a*, *b* and *c*, pass through the nodal points of the lens and appear on the retina at the points *a′*, *b′* and *c′*. They are projected out into space by the brain along the lines by which they enter the eye. Point *b* enters the eye, and without any deviation is reflected along the same line to *b* in space. Points outside the center of fixation, as *a* and *b*, enter the eye obliquely, and are projected along the same oblique line of entry. There is a definite relation, therefore, between *b* and *b′*, *a* and *a′* and *c* and *c′*. What is true of these points is equally true of all other points in space in relation to fixed points in the retina when the eye is at rest.

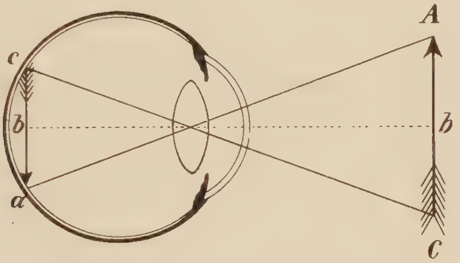

Fig. 40.—Laws of projection and direction.

It will be noted in the diagram that objects above the point of fixation fall upon the lower part of the retina and *vice versa*. Similarly, points in space from the right of fixation fall on the retina to the left of the macula, and objects from the left fall on the right of the macula.

Law of Corresponding Retinal Points.—The fundamental laws just discussed concern one eye. Normally, the eyes do not operate singly, but function together. In the anatomy of the visual pathway, the student may recall that the right halves and the left halves of the retinæ are known respectively as corresponding, or homonymous, retinal halves, and that these corresponding halves receive impressions which are recorded in the cerebral cortex as a single impression. In order that they may function as one, higher cerebral centers in the mid-frontal convolutions of the brain, direct conjugate movements to the right and left, up and down, etc. In normal individuals with normal muscle balance and normal fusion power, bilateral foveal fixation results in single vision. The two foveæ, therefore, are known as corresponding retinal points. If points on the horizontal meridians of the retinæ are selected, for example, points 5 degrees to the right side of the maculæ, an object in space to the left of fixation which falls on these points will be seen as one. They are, therefore, called corresponding retinal points. Every point in the retina of one eye has a corresponding retinal point in the other and, if the eyes are properly conjugated and coördinated, single vision will result although the object is perceived in each eye. This law of corresponding retinal points brings not only definite points into intimate relation, but retinal halves and retinal quadrants as well.

Physiological Diplopia.—If the reader will hold directly in front of him two pencils, one at 6 inches and the other at 12 inches distance, and will fix his vision on the far pencil, he will notice that the proximal pencil will be seen as two, separated about 1 inch. If he will now alternately close one eye and then the other, he will note that the left image belongs to the right eye and the right image will be seen by the left one. The condition is crossed diplopia. If he will now reverse the experiment and will fix on the near pencil he will observe two pencils in the distance, the right image belonging to the right eye and the left one to the left eye uncrossed or homonymous diplopia. If we were always to recognize an object in indirect vision as two we would be hopelessly confused. According to Tscherning, one image is suppressed, the right-hand image appearing more real and substantial in right-handed people and *vice versa* in left-handed people. Physiological diplopia is of much value in measuring depth in binocular vision.

Binocular Vision.—Binocular single vision is normal in the human race. It is made possible by: (1) The partial decussation of the retinal fibers in the chiasm, so that the brain can receive impressions from corresponding retinal points as a single impression; (2) by means of cerebral centers governing conjugate movements, so that the eyes may at all times be placed to receive impressions on corresponding retinal points; (3) by reason of a fusion faculty, which enables the individual to superimpose his foveal images and make them one.

Evolution of Binocular Vision.—A study of the evolution of binocular vision is of much interest. In the lowest orders of animal life the eyes are placed in the sides of the head. There is no overlapping of the visual fields. The eyes operate independently. In ascending the scale of the animal kingdom the eyes gradually move forward and the visual fields begin to overlap. There now is a tendency to concerted action. In the higher orders the visual axes tend to approach parallelism. Only in apes and in man, however, does parallelism reach a reality. With parallelism of the axes, binocular vision not only becomes possible, but essential.

In the human embryo a gradual evolution of the position of the eyes from a lateral to a frontal position in the head is observed. At birth the infant's eyes are potentially parallel, but there is no coördination of the lateral ocular movements. As a rule, the vertical balance is normal, but for several weeks the eyes may be divergent or convergent. Gradually the infant begins to note bright objects. From the sixth to the eighth week its attention can be fixed upon a bright object and its attention may be held. The mother says "The child sees," a condition which the psychologist calls "the unfolding of the faculty of attention." We call it the development of the fusion faculty. At the end of three months, if normal, this faculty is well developed. An occasional squint may be observed, but with succeeding months fusion grows in strength. At the end of the year the following prism experiment may be practised with success. A 3 or 4 diopter prism, base in, may be placed before one eye when the baby's attention is fixed and the usual compensatory movement of the eye before which the prism is placed will be noted. If fusion is not well developed no movement will be observed. From clinical experience we have observed that fusion training yields encouraging results up to the seventh year. After this period results are less satisfactory. This is a clinical fact of much importance. It argues for the setting into motion measures to correct squint before the child reaches its seventh year, a practice which is only too often neglected in the ordinary routine of clinical practice.

Binocular Fixation, Binocular Single Vision and Stereoscopic Vision.—Binocular single vision is as much a function of corresponding points in the peripheral retinæ as it is of the foveæ. In the fixation of objects, however, corresponding retinal points other than the foveæ are for the moment disregarded. It is evident from this fact alone that there is some difference between binocular fixation and binocular single vision. As pointed out by Maddox, binocular fixation is a subconscious act, automatic so to speak. It becomes the result of habit. Binocular single vision implies more. It engages the other parts of both retinæ in exactly a similar manner and yet these "points of attention" to which the mind is invited in the peripheral retinæ require a more or less conscious act on the part of the individual in their recognition. For the complete and perfect interpretation of objects seen or observed in binocular vision there is another phase which needs consideration, namely, stereoscopic vision or the preception of depth. An individual may possess a fair degree of the faculty of binocular fixation and binocular single vision, and yet may have little power of stereoscopic vision. For perfect binocular vision in its truest sense, vision which embodies these three components, a fusion faculty is essential.

Fusion.—In a study of binocular single vision, inasmuch as an object which falls on corresponding retinal points is received as a single impression in the cortical visual center, one might on first thought regard the act as a simple automatic act, which needs no further explanation. That it is more than a simple automatic act is best demonstrated by a prism experiment. If, during this act a 4 diopter prism is placed before one eye, base in, the discomfort caused by the displacement of the image of this eye, is followed unconsciously by a corresponding rotation of the eye outward and singleness of vision is restored. There is then a desire or a love for single vision. It is not a purely automatic act, but an active definite effort toward a definite end. This desire for singleness of vision we call a fusion faculty. By some the abhorrence of double images is thought to be the force which impels toward single vision. Van der Hoeve, therefore, coined the phrase "diplopia phobia" as expressive of the condition. This negative way of regarding the phenomenon but implies that there should be a positive force or ability to overcome diplopia. It is more expressive of the real condition to speak of a love for or a desire for fusion. Whether we call the force which impels to fusion a "fusion faculty" or a "fusion center" is purely an academic question with the weight of the argument in favor of the former. We know of no such center and it is safe, therefore, to speak of a "fusion faculty." It is noncommittal. We will regard it, therefore, as a positive force which impels, when possible, to single vision.

Breadth of Fusion.—When prisms are placed before one eye during fixation the lower the prism, and, naturally, the less the displacement, the easier it is to overcome the diplopia. That is, the greater apparently is the desire for fusion. If the prism employed is too large fusion becomes impossible. The breadth of fusion varies with the direction in which the apex of the prism is placed. A prism 3 to 5 diopters placed apex up or down will rarely be overcome. The power of overcoming vertical displacements is slight. In the lateral, a prism of about 8 diopters is about the maximum which can be overcome if placed base in. The power of overcoming high degrees, base out, varies with the individual, but this faculty may be developed in most individuals to a very considerable extent, even to 70, 80 and 90 diopters.

In order to measure the breadth of fusion in the vertical, the strongest prism base down is added to the strongest prism base up which can be overcome. The lateral breadth is the sum of the strongest prism base out and the strongest prism base in which can be overcome. There is comparatively little variation in the vertical in different individuals, but in the lateral the variation is great.

From the clinical standpoint, Maddox suggests three groups: (1) Those in whom the breadth of diplopia is greater than that of fusion. Fusion is impossible. This condition is found in permanent concomitant squint. In the second group the breadth of diplopia and of fusion are nearly equal, and the diplopia may be overcome by effort. This occurs in periodic squint. In the third group the breadth of fusion is greater than the diplopia. This condition occurs in heterophoria or latent squint. A fourth group should be added, namely, a normal group, in whom the breadth of fusion is always sufficient to maintain single binocular vision.

Estimation of Depth by Monocular and Binocular Vision.—Although the third dimension is better perceived binocularly, it is possible to obtain a fair measure of the solidity of an object or a sense of depth by monocular vision. Both forms of vision are assisted by the same factors, the greater efficiency of binocular vision being due largely to convergence. Of the factors which are common to both, the more important ones are as follows: (1) Atmospheric density; distant objects are more or less veiled because of atmospheric density; in mountainous districts the atmosphere is relatively clearer and very distant objects seem to be nearer than they virtually are; when the atmosphere is heavy, as in a fog, objects seem to be further off than in reality. (2) The relative visual angle of known objects; the size of a known object such as a man, a horse, etc., when seen at a distance, furnishes fairly accurate data upon which to measure the intervening distance between the object and the observer. (3) Shadows thrown upon an object, if the source of light is in the rear, at once gives one the impression that the object casting the shadow is nearer to the observer; this is especially helpful if the intervening objects are well known as to size and dimensions. (4) The perspective obtained by the seeming approach of parallel lines, such, for example, as obtained by gazing down a street, is a most valuable aid in measuring depth.

(5) The effort of accommodation is of some value, perhaps more in the estimation of depth very near at hand, because accommodation is only operative at close range. (6) The parallax is most important, but obtained in a slightly different manner in monocular than in binocular vision. In binocular vision the eyes view an object at slightly different angles; depth and solidity are easily obtained; a single eye accomplishes the same object if the head is shifted from one side to the other; the results obtained are good in either case, but the latter requires a more conscious effort than the former. (7) The most important factor in the estimation of depth and a function of binocular vision only is that of convergence. The special value of this last aid in perceiving depth lies in the difference in the converging power required in passing from one object to another. Thoroughly mixed, however, with this act is the role of physiological diplopia which enables or leads one from a definite point of fixation to the images seen in physiological diplopia. This combination, therefore, of relaxation and readjustment of convergence, and the shifting of fixation alternately on a far and a near point, furnishes the real stereoscopic vision in which objects stand out in relief.

The Stereoscope.—When one views an object in relief, a landscape scene, for example, an image of this scene appears before each retina, but at slightly different angles. They are exactly like the two pictures taken in a stereoscopic camera, which differ only by the slight angle which is formed by the base separation of the two camera plates. When two photographs taken by two camera plates, separated probably about the same distance as the human eyes, are so projected that they can be fused into one, the observer obtains the same effect as he obtains by looking upon the scene with his own eyes, provided he has true stereoscopic vision. In 1883 Wheatstone gave to the world his stereoscope, which consisted of mirrors at right angles. This is illustrated in Fig. 41. This form of stereoscope is now an important part of a complete Roentgen-ray outfit, in which stereoscopic vision plays an important role.

The value of the stereoscope is best appreciated by the great number of models which have been placed on the market from time to time and the ever increasing number which is being added, one might say yearly. In 1849 Brewster offered an improvement in which lenses were added, which when properly decentered produce the effect of prisms, bases out, thus enabling the observer to project the two pictures or images to the same spot, giving one the effect of relief (Fig. 42). At the same time the lenses slightly magnify and the illusion, as Hansell and Reber point out, is complete. This particular form or model has undergone many changes. The most important models which have been produced for a specific purpose and have endured are the Holmes models, now usually seen in the shops, Mr. Berry's model, the Worth-Black amblyoscope, a model constructed by Wells for training in heterophoria, and the Lloyd stereoscopic slate for perimetric purposes.

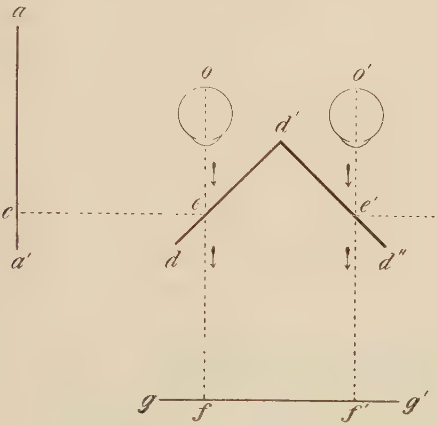

FIG. 41.—Wheatstone's stereoscope. (From "Ocular Muscles," by Hansell and Reber.)

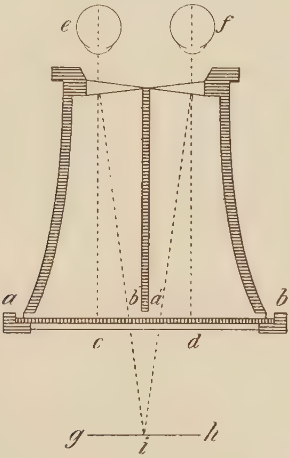

FIG. 42.—Brewster's stereoscope. The pictures *c* and *d* are blended or fused in the plane *g*—*h*. (From "Ocular Muscles," by Hansell and Reber.)

Mr. Berry's addition to the stereoscope is of much value (Fig. 43). A circle is placed before each eye, and in the center of each circle is placed a smaller circle which can be displaced from side to side. When the small circles are displaced centrally, *i. e.*, away from the centers of the large circles toward each other, the sensation obtained is one of relief. It gives one the appearance of a tub upside down. It corresponds to the object in the foreground of the picture. If the inner circles are now displaced toward the outer edges of the large circles, it is as though one were looking into depth. The tub is now righted and one looks into it. This corresponds to the objects in distance and conveys the sensation of depth. To the value of this test for the determining of the presence of true stereoscopic vision, the author can bear testimony. Lloyd has taken advantage of the principle in securing perfect fixation on his stereoscopic slate.

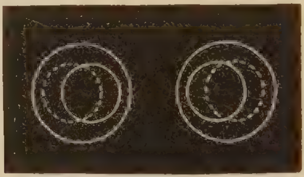

Fig. 43.—Mr. Berry's stereoscope. (From Maddox's "Ocular Muscles.")

Worth-Black Amblyoscope.—The use of the stereoscope in squint is limited because the angle of squint does not permit the images to approach sufficiently near for the fusion faculty to exercise its influence. Claude Worth, therefore, constructed a stereoscope, which essentially consists of Priestly Smith's fusion tubes fastened by a hinge, so that the angle of deviation of the squint can be approximated, and the images may be brought close enough for the operation of the fusion faculty, if it is present. In some of these cases of squint one eye may be more or less amblyopic or vision may be subnormal because of long suppression of the image (Fig. 44). Black conceived of the valuable addition of a rheostat, whereby one may increase the intensity of the illumination in one tube and diminish that of the other. This has made of the Worth-Black amblyoscope a most excellent instrument for the training of squint—a procedure which is of incalculable value.

Tests for Binocular Vision.—In making tests for binocular vision one must clearly differentiate between binocular vision without any or only a moderate degree of depth perception and true stereoscopic vision. For example, the letter *L* may be placed before one eye in a stereoscope or amblyoscope and the letter *F* before the other eye. In simple binocular vision these letters may be blended into an *E*. This does not guarantee stereoscopic vision. If, however, the principle of Mr. Berry's stereoscopic chart, or a modification of it, is employed one can determine with absolute certainty that true stereoscopic vision is or is not present. This principle can be applied to very young children, and they can tell with assurance as to whether they are looking into a bucket or at the bottom of a bucket upside-down. In the author's experience this is the most valuable test at our disposal. It can be varied so much that the answers leave no doubt as to their accuracy.

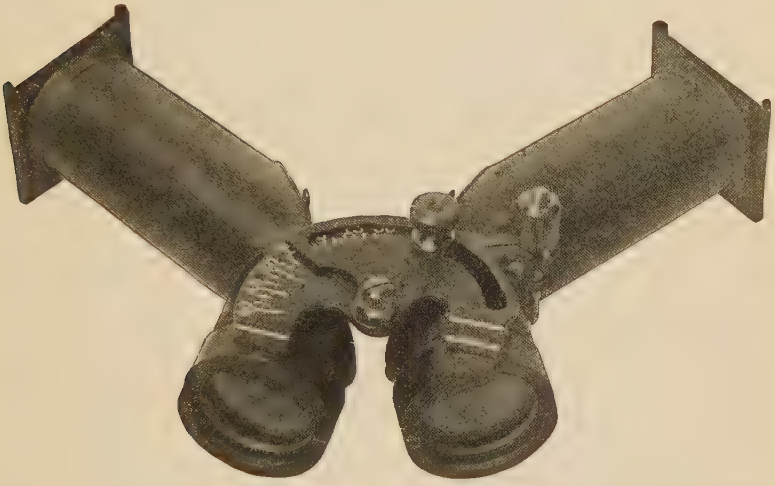

Fig. 44.—The Worth-Black amblyoscope. (From deSchweinitz's "Diseases of the Eye.")

The Bishop Harmon diaphragm test (Fig. 45) is of considerable practical value. It also is a good test for malingering. In this test the charts are placed in the carrier at the extreme end of the instrument, and the diaphragm can be adjusted with such nicety that the extreme left of the card can only be read by the right eye and the extreme right by the left eye. The center of the card is read by both eyes. If this card can be read easily, without moving the instrument from side to side, there can be no question as to the presence of binocular vision. It does not, however, determine the presence of stereoscopic vision.

Bar reading has the same objection. If a ruler, for example, is held between the patient's eye and a book the patient may be able to read, as in the Bishop Harmon diaphragm test, without difficulty provided binocular vision is present. If not carefully watched, the patient may move his head slightly and continue to read with one eye. Furthermore the eyes may be used alternately when the obstruction is encountered.

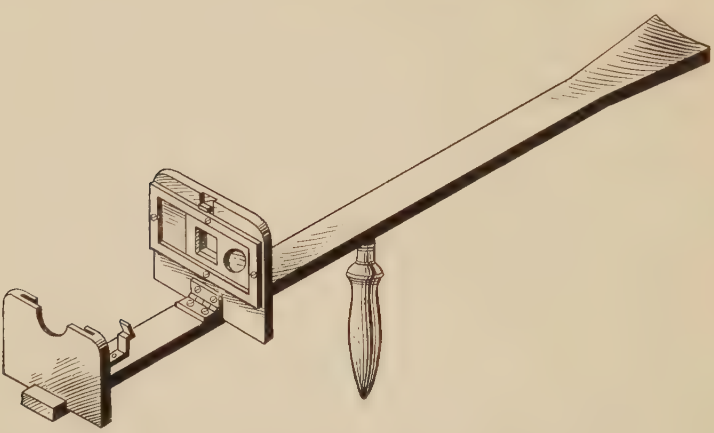

FIG. 45.—Bishop Harman's diaphragm test. (From "Ocular Muscles," by Hansell and Reber.)

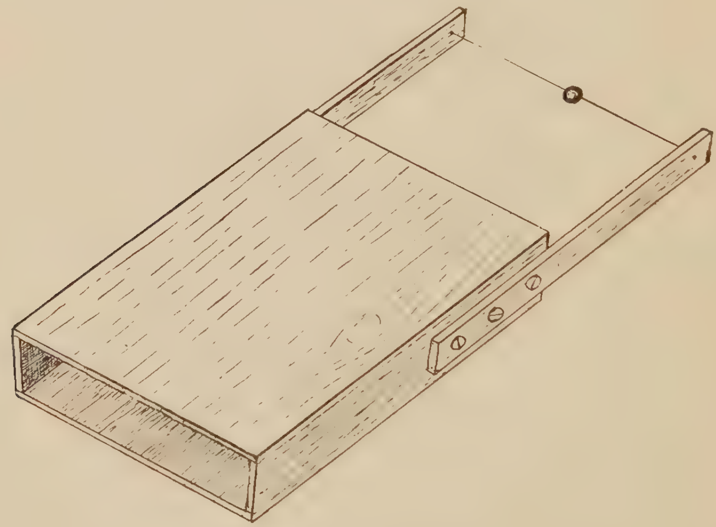

FIG. 46.—Hering's drop test for the sense of perception. (Courtesy of Worth and Baillière, Tyndall and Cox.)

The Hering drop test is said to be of satisfactory merit. It is not, however, infallible, although usually dependable. It may be constructed out of a flat cigar box from which the ends have been removed. To one end two arms are projected, as shown in Fig. 46. A thread is stretched across the distal ends of these projecting arms, on the center of which a small bead is placed and upon which the patient may fix. Small round objects like marbles, dried peas, etc., are now dropped between the thread and the box. Care should be observed that the balls passed beyond the thread should be a little larger than those passed inside the thread. If the patient has true binocular vision he should be able to tell on which side of the thread the balls are dropped. The test, however, is not without error.[1]

Contrary to the generally accepted belief that the Hering drop test is of greatest value, the author believes that the stereoscope or the amblyoscope is more refined and accurate in determining the degree of true single binocular vision if present.

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[1] Worth believes this test to be one of "subconscious perception of physiological diplopia in more or less eccentric parts of the retinæ."

PART II.

HETEROPHORIA.

General Considerations.—Heterophoria is a general term which includes all of the various forms of latent tendencies to deviation from a normal muscle balance. Earlier in our discussion, reference was made to the state of the extra-ocular muscles during consciousness and unconsciousness. During unconscious moments the eyes are rarely found in a state of parallelism, but usually deviate upward and outward. This was called position of *anatomical rest*. The position is determined by the shape of the orbits, the attachment of the optic nerve and possibly in part by the elasticity of the check ligaments and orbital fascia in general, the ocular muscles being in a state of relaxation. When consciousness returns, without effort, the position is changed from that of *anatomical rest* to *functional rest*. *Functional*, because from this position of "attention" the eyes are ready to respond to any innervational influence which may present itself, and *rest* because the unconscious innervation, which brings the eyes to "attention," requires no effort. Although potentially present, heterophoria or latent squint does not become operative until innervational influence demands conscious effort.

When heterophoria is present it tends to incoördination which brings about the symptoms which so often baffle even the most experienced ophthalmologist. It is not, however, the symptoms to which the incoördination gives rise, which cause the greatest anxiety. The cause of the symptoms is easily determined by routine methods of study. The reasons why heterophoria should exist in a given patient furnish the real difficulty in the case. Is it a refractive error alone, as some would have us believe? The relation of accommodation to convergence, even in an emmetrope, is so intimate and close that one, without giving the subject much thought, could be induced to believe that the influence of refractive errors might be all sufficient to bring about the incoördination. This, however, is only a part of the story—an important phase, indeed, but others equally vital must be considered.

There is, it is true, a type of heterophoria which may be called accommodative. For example, a patient suffering from 3 diopters of hypermetropia may show a tendency to increased convergence during the act of accommodation, which may entirely disappear if the hypermetropia is corrected. In a similar manner myopia, if uncorrected, may be accompanied by exophoria. This, too, may be overcome by the wearing of a suitable correction. This group of heterophorics is in the minority.

Other factors enter into the production of disturbed muscle balance. Individual muscles may show some abnormality in attachment, or may be overdeveloped as compared with the antagonists. Occasionally there is present an anomalous condition which may be a direct cause. In vertical disturbances the motor factors just mentioned may be the direct cause of an over- or an underaction in response to an innervation. If these can be excluded, and even when present, the investigation of heterophoria must go further.

What is the patient's temperament, his physique, his occupation, his state of health, his hours of occupation, his habits of worry and, above all, his heredity, are subjects which need the closest scrutiny. While it is difficult to understand all that is involved in the expression "innervations," it is not hard to comprehend how difficult and intricate the mechanism is which brings about response to these influences. Nor is it difficult to conceive how easily a mechanism, which involves the fine coördination of at least twelve delicate ocular muscles, can be influenced and disturbed by one's temperament, physique, state of health, occupation, fatigue and a host of other conditions to which we have not alluded. A study of the principles involved and the treatment of the symptoms or clinical manifestations resulting therefrom, offer to the ophthalmic surgeon an opportunity for the exercise of rare judgment as a physician as well as profound knowledge of the subject of refraction. In dealing with squint, whether concomitant or paralytic in character, we are confronted with fairly definite facts which follow rather definite laws. In heterophoria, on the other hand, each case becomes an individual study, and the results obtained, in giving relief to the suffering of its victim, depend largely upon a thorough comprehension of the factors which generate it and the multitude of symptoms which attend its presence.

It is well to recall at this time that the ocular muscles never act as individual muscles, but that each movement is the result of the combined action of synergists, with corresponding relaxation of opposing groups. A tendency to insufficiency of convergence, for example, should not be imputed to weak internal recti, nor should excessive convergence be thought of as overaction of the interni. It happens, not infrequently, that anomalous attachments and abnormal developments of single muscles may be a factor, but it is impossible to disassociate the internal rectus, for example, from the action of the vertical recti. Lancaster[1] recently emphasized the fact that each ocular muscle possesses more power than is needed for any act which it is called upon to do. He concludes with entire correctness, that the deficiencies encountered are, therefore, largely innervational.

Two other facts, common to all forms of heterophoria, need stressing in general considerations. The first is this: Although the tendency to squint is present, the patient can hold it in check by effort. It is the *effort* which is largely responsible for the symptoms which follow. When the patient is no longer able to hold the balance, manifest squint appears and the entire disassociation of the two eyes brings relief. Symptoms, other than the manifest squint, now disappear. A second fact of importance is the difference in reactions of different individuals to similar forms of muscular incoördination. Hyperphoria, vertical disturbance, which is responsible for so much discomfort to the average patient may not cause any inconvenience to others. This may be because of inherent differences in the general make-up of the individuals. On the other hand, the same patient may find close work impossible, and may enjoy perfect comfort and well-being when following an out-of-door life, although the same degree of heterophoria may be present for distance and for the near-point.

Nomenclature. The nomenclature suggested by Stevens for functional deviations has been generally accepted in America and abroad. It is as follows:

Orthophoria.—Perfect binocular muscle balance.

Heterophoria.—A tendency to imperfect binocular muscle balance. This is a general term which includes the following subdivisions:

Hyperphoria.—A tendency of the visual axis of one eye to deviate above that of the other. It is designated as right or left hyperphoria.

Hypophoria. A tendency for the visual axis of one eye to deviate below the other. It may be right or left. This term is not much used, as the vertical deviation is usually expressed in terms of hyperphoria, right or left. As it is possible to determine in some cases that one visual axis is too low rather than the other too high, hypophoria should have a place in our nomenclature. Savage suggested the term *cataphoria* to indicate this condition. The author, however, is inclined to believe that harmony will be preserved if the term *hypophoria* is used to express the opposite condition from that of *hyperphoria*.

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[1] South. Med. Jour. Jour., 1924.

Double Hyperphoria.—This is a term applied to indicate the elevation of both visual axes. Stevens called this condition *anaphoria*. If double hyperphoria is used it will obviate the necessity for the addition of another word to our nomenclature.

Double Hypophoria.—This signifies lowering of both visual axes below the projected horizontal plane of the head. For this condition Stevens used the term *kataphoria*. Like anaphoria it does not simplify, but adds additional words to our nomenclature. In the interests, therefore, of simplicity and accuracy, it would seem best to drop the terms *anaphoria* and *kataphoria*, as suggested by Stevens, and *cataphoria*, as suggested by Savage, to indicate the lowering of the axis of one eye below the horizontal plane of the head.

Esophoria.—A tendency for the visual axes to deviate inward.

Exophoria.—A tendency for the visual axes to deviate outward.

These terms may be compounded into *hyper-exophoria*, a tendency to deviation upward and outward, and *hyper-esophoria*, a tendency to deviation upward and inward. To be correct, one should designate the hyperphoria as right or left, although the right and left are not applied to exophoria and esophoria. In the author's judgment, the terms should be used separately, as, for example, exophoria with right hyperphoria, etc.

Cyclophoria.—This is a term contributed by George Price, and indicates a tendency of the upper end of the vertical meridian of the cornea to deviate out or in. To this condition Stevens gives the name of *declination*. Cyclophoria is the generally accepted term.

Hyperkinesis.—This, according to Duane, indicates excessive action of an individual muscle.

Hypokinesis.—Deficiency of action of an individual muscle. (Duane).

Parakinesis.—Irregular action of an individual muscle. (Duane.)

Logically, although the three terms mentioned last are expressive of the conditions as indicated, they should not be included under the nomenclature of heterophoria, since they refer to individual muscles rather than to groups of muscles which respond to innervations.

PRISMS.

In the diagnosis and treatment of heterophoria, as well as in manifest squint, the use of prisms is most essential. It is important, therefore, that the properties of prisms and their application in the functional disturbances of the ocular muscles be considered at this time.

A prism differs from ordinary glass, such, for example, as a window pane, in that the two surfaces, instead of being parallel to each other, are inclined at an angle. This inclination forms an angle, also called the apex or edge of the prism. When a ray of light, therefore, passes through a prism, instead of passing through without bending or refraction, as it would through a window glass, it is bent from its course and is said to be refracted toward its diverging edge or the base of the prism. It is a law of physics that a ray of light passing from a rarer to a denser medium is deflected toward the perpendicular, and when it passes from a denser to a rarer medium it is bent away from the perpendicular. In Fig. 47 a ray of light from *A* which is made to pass through a prism placed before the eye is deflected toward the perpendicular of the prismatic surface, *x y*, because the glass is denser than air. As it leaves the second surface of the prism, *x z*, it is bent away from the perpendicular and enters the eye. The light, *A*, therefore, is seen, not at *A*, but as though displaced toward the apex and virtually appears as at *A′*. A prism, therefore, is said to refract toward its base and to displace the image of an object toward the apex, angle or edge.

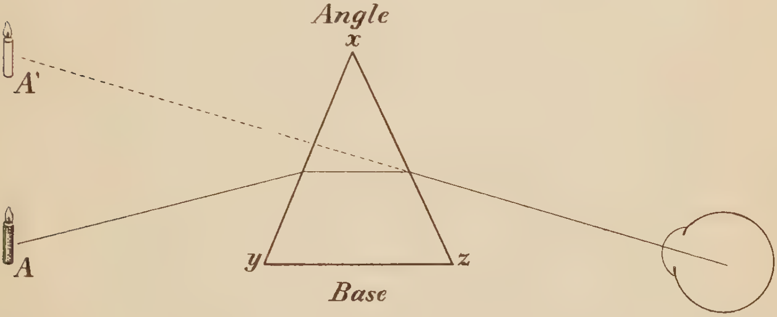

FIG. 47.—Action of a prism on a beam of light. Dotted line indicates direction which the projected beam takes.

Numbering of Prisms.—Prisms were formerly numbered by the refracting angle formed between their two refracting surfaces. They were marked from 1 degree up. Thus designated, the numerals simply indicated the angles of the refracting surfaces and did not express the amount of deviation which a ray of light would undergo in passing through a prism so designated. To Dr. Edward Jackson we are indebted for his bringing the subject before the Ninth International Congress, when a standard nomenclature was adopted and prisms were designated by their power of deviation.

Two methods were proposed, now universally adopted, one by Dennett and the other by Prentice.

Dennett's Method.—In this method the centrad is the unit, designated symbolically by the Greek letter delta inverted, ▽. A 1 centrad prism will deviate a ray of light the 0.01 part of the arc of the radian. This is arc measurement and is equal to 0.57295 degrees. The value of the centrad is obtained as follows: In Fig. 48 *A* and *C* is the radian of the arc which is obtained by measuring off on the circumference of the circle a distance equal to the length of the radius of the arc, *A R*. The radian is equivalent to 57.295 degrees. The centrad is 0.01 part of the radian, or 0.57295 degrees. This unit of measure is constant and the value of any multiple of the unit, as, for instance, 10 centrads, is obtained by multiplying the value of the unit 0.57295 degrees by 10. The value of 10 centrads is 5.7295 degrees.

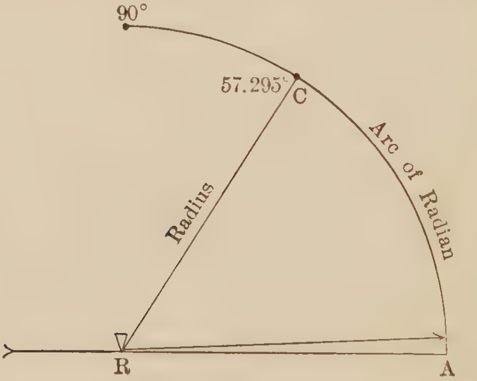

Fig. 48.—Illustrating Dennett's method of numbering prisms. (From Thorington's "Prisms.")

Prentice's Method. The unit of measure adopted for this method is called prism diopter and symbolically is designated by the Greek letter delta, △. One prism diopter will deviate a ray of light 1 cm. for each meter of distance. At 2 meters the deviation will be 2 cm., etc. It will be noted in Fig. 49 that the prism diopter is tangential measurement in contrast to the arc measurement of the centrad. The two units of measure are so nearly equal in value up to 20 that either system may be used with equal accuracy. Beyond 20 centrads or 20 prism diopters, the centrad continues to be uniform while the prism diopter loses in value. (See appended table.)

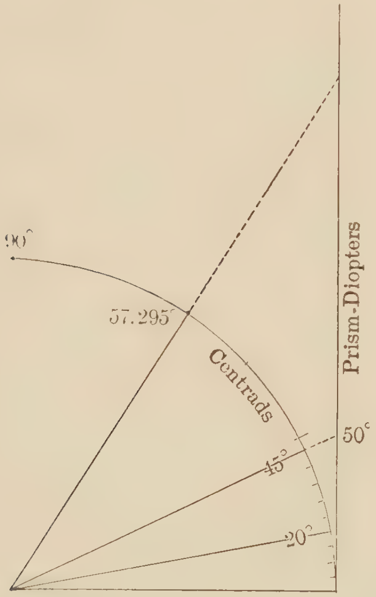

FIG. 49.—Comparing Dennett's and Prentice's methods of numbering prisms. (From Thorington's "Prisms.")

Centrads.	Prism-diopters.	Refracting angle.	Centrads.	Prism-diopters.	Refracting angle.
1	1.0000	1° 00	17	17.164	16°.98
2	2.0001	2°.12	18	18.196	17°.85
3	3.0013	3°.18	19	19.230	18°.68
4	4.0028	4°.23	20	20.270	19°.45
5	5.0045	5°.28	25	25.550	23°.43
6	6.0063	6°.32	30	30.934	26°.81
7	7.0115	7° 35	35	36.500	29°.72
8	8.0172	8°.38	40	42.280	32°.18
9	9.0244	9°.39	45	48.300	34°.20
10	10.0330	10°.39	50	54.514	35°.94
11	11.0440	11°.37	60	68.430	38°.31
12	12.0570	12°.34	70	84.220	39°.73
13	13.0740	13°.29	80	102 960	40°.29
14	14.0920	14°.23	90	126.010	40° 49
15	15.1140	15°.16	100	155.750	39° 14
16	16.1380	16° 08			

Relative value of centrads and prism diopters. (Courtesy of Thorington and Publishers.)

RELATION BETWEEN ACCOMMODATION AND CONVERGENCE.

To Donders we are indebted for concise knowledge of accommodation and its unit of measure, the diopter of accommodation, and to Nagel for the unit of measure of convergence, the meter angle of convergence. The relation of these units is most vital in a study of heterophoria. It is well known that if the ciliary muscle of an emmetropic eye is completely controlled by a cycloplegic, such as atropine, the patient when looking at infinity will not be able nor will he find it necessary to call into play any accommodative power in order to see clearly. When we substitute for infinity the usual testing distance on the Snellen chart, 6 meters, the same condition obtains. If, on the other hand, he is asked to read the letters which the normal emmetropic eye, without cycloplegia, should read at 1 meter's distance, he will be unable to read unless a 1 diopter sphere is placed before the eye. He is said, therefore, to use 1 diopter of accommodation. Similarly, at 0.5 meter's distance, or 50 cm., he will need a 2 diopter sphere, which is equivalent to 2 diopters of accommodation. At 33 cm. he will accommodate 3 diopters, and at 25 cm. he will require 4 diopters of accommodation, etc. These are definite and fixed quantities of accommodation required by a normal emmetropic eye.

As the patient normally uses both eyes in the act of accommodation, it becomes necessary for the eyes to converge in order to fix on any point nearer than infinity. To correspond to the diopter of accommodation required by an emmetrope to read at 1 meter's distance, Nagel expressed the degree of convergence required of each eye for this act as a *meter angle of convergence.* At 50 cm. the convergence will amount to 2 meter angles; at 33 cm., 3 meter angles; at 25 cm., 4 meter angles, etc. It will be observed, therefore, that by this facile mode of expressing the degree of accommodation and the degree of convergence, a normal relation is clearly established and the disturbed relation in ametropia is easily followed.

Effect of Ametropia on this Relation.—Let us assume an error of 2 diopters of hypermetropia. In order to read Snellen type at 1 meter, this patient will require not only the normal 1 diopter of accommodation, but must add on his 2 diopters of spheric error, making a total of 3 diopters. For this same distance he will, however, require but 1 meter angle of convergence. At 50 cm. he will require 2 diopters of accommodation normally plus 2 required by his ametropia, making a total of 4 diopters of accommodation to 2 meter angles of convergence. At 33 cm. the relation will be 5 diopters of accommodation and 3 meter angles of convergence. At 25 cm., 6 diopters of accommodation and 4 meter angles of convergence, etc.

If the patient's ametropia amounts to —2 diopters the relations will be as follows: At 1 meter no accommodation will be required, but 1 meter angle of convergence will be necessary; at 50 cm., no accommodation, 2 meter angles of convergence; at 33 cm., 1 diopter of accommodation will be necessary and 3 meter angles of convergence. At 25 cm., 3 diopters of accommodation will be called into action and 4 meter angles of convergence.

These disturbed relations are graphically expressed in the following table:

	Emmetropia.		+ 2 D.		− 2 D.	
	A.	C.	A.	C.	A.	C.
6 meters	0	0	2	0	0	0
1 meter	1	1	3	1	0	1
50 cm.	2	2	4	2	0	2
33 "	3	3	5	3	1	3
25 "	4	4	6	4	2	4

The vital bearing which disturbance of the relation between accommodation and convergence, caused by ametropia, will become more apparent in the discussion of esophoria and exophoria. At this point, however, the reader will experience no difficulty in noting that hypermetropia tends to increase convergence and that myopia, by the lowered accommodation required, tends to bring about defective convergence. It is upon these tendencies that some believe the "accommodative theory" of squint furnishes a satisfactory working basis by which to explain the etiology of squint. Final judgment, however, should be held in abeyance until the etiology of squint is more thoroughly discussed.

A point of practical importance, which the disturbance of accommodation and convergence in ametropia suggests is the "drawing" or "pulling," of which many patients complain when wearing glasses for the first time, if the correction is too full. The forced breaking-up of a relation between accommodation and convergence, which is not normal, but which was acquired during the years when glasses were not worn, often results in discomfort to the patient aside from the blurring which follows failure of the ciliary muscle to relax after a full correction is ordered. It is an argument against prescribing glasses under a cycloplegic as a routine matter or the routine "rule of thumb" cutting of the sphere as applicable to every case—a practice which is only too often followed in every-day refraction.

MUSCULAR DYNAMICS.

In studying heterophoria it is essential that we investigate the rotating power of individual muscles, with their accompanying synergists or associates, of each eye separately and the ability to overcome prisms when the two eyes are used together. The first is a test of muscle strength or ability to respond to an innervation with one eye occluded, and refers to the rotating power of an eye. The second is a study of prism duction power, a test of the patient's fusion power. The two are often confounded. In earlier sections an effort was made to pave the way for a better differentiation of these two functions of the eyes, by limiting the terminology in rotations to external, internal, outward, or inward, temporal or nasal, etc., and the terms abduction, adduction, supraduction and infraduction were not employed. These terms have been reserved to express the ability of the eyes to overcome prisms. It may be argued by some that this is drawing too fine a distinction, but it is the author's conviction that if carefully followed it will help the student to think more clearly and concisely, and the difference between rotations and ductions will always be definite.

Rotations.—The subject of arc rotations was discussed under "fixation fields" and the methods were fully described. These studies are of greater importance in squint. In all complicated cases of heterophoria, however, the rotating power of each eye may be of much help in arriving at the best method of treatment of the heterophoria.

Prism Duction Power.—In a study of heterophoria, and especially in the treatment of the same, it is not only necessary to determine the kind and degree of the heterophoria, but a knowledge of the duction power is equally essential. For example, in exophoria it is necessary to know the strength of the abducting and adducting power of the patient, because on the relation of these will depend the treatment to be prescribed to correct the exophoria.

Ductions are obtained by placing increasing strengths of prisms up or down, in or out, before the eye, as the case may be, until the displacement of the images becomes insuperable and manifest diplopia appears. The tests should be made at a distance of 6 meters from the muscle light, in a slightly darkened room, the light being on a level with the patient's eyes or a little below this level.

Supraduction. In order to test supraduction a 1 diopter or centrad prism should be placed in a trial frame before the right eye, for example, base down, and the patient is asked to watch the muscle light carefully for the appearance of a second image displaced upward. It should be recalled that the image is displaced toward the apex of the prism. The light will, therefore, fall below the macula. If the patient's power for fusion is normal the macula will be rotated downward, in the interests of single binocular vision, and the two lights will be seen singly. A downward rotation of the macula is accomplished by rotating the cornea of the right eye upward, hence the term supraduction. The strength of the prism is gradually increased until the patient is no longer able to fuse the two images into one if they have separated. The strength of prism which the patient is able to overcome and maintain single binocular vision is a measure of the supraduction. If the patient can overcome the displacing effect of a 3 diopter prism and 4 diopters produce insurmountable diplopia, the measure of the supraduction is 3 diopters. The normal average supraducting power is 2 to 3 diopters. These are rather constant values. Few patients can overcome more than 4 diopters with the prism placed base down. A recent patient of the author's was able to fuse 8 prism diopters displacement. This is exceptional.

It is immaterial whether the prism is placed before the right or left eye. The results should be the same or approximately the same in average eyes. The test is not one of muscle strength, but a test of the ability of eyes to operate disassociatedly in the interests of single binocular vision. The reader may recall that a vertical balance is present in an infant at birth. It also is a clinical fact, recognized by all, that in the treatment of hyperphoria little can be accomplished by exercising the vertical balance by means of prisms. The test is a binocular one and should be the same whether the prism is placed before the right or left eye. In those cases in which a right supraduction is found greater or less than when the prism is placed base down before the left eye the discrepancy may be due to: (1) A lack of proper care on the part of the patient or operator; (2) the slight influence of exercise or perhaps greater effort on the part of the patient as the tests proceed; and (3) the influence of fatigue. Note that one eye does the fixing.

Infraduction.—Like supraduction, infraduction, as a rule, is between 2 and 4 diopters. It is measured by means of prisms placed base up before either eye. It is slightly in excess of supraduction. This also is influenced by fatigue. If infraduction is measured immediately after supraduction is taken, the measurement may be slightly less than if taken first. The reverse, in the author's experience, shows greater variation. That is, if supraduction is taken immediately after infraduction is measured, the measurements of supraduction are more apt to show minimum duction.

The sum total of supraduction and infraduction is the amplitude of vertical ductions. The amplitude in the vertical falls far below that of the lateral, a fact which will have great clinical significance in the correction of hyperphoria.

Abduction.—The ability to overcome prisms, placed base in before one or both eyes, is known as abduction. The process by which this is accomplished is as follows: When a prism is placed base in before the right eye, the left eye fixing on the muscle light at 6 meters, the prism displaces the image of the light before the right eye toward the apex or angle. The image of the light will, therefore, fall to the inner side of the macula. In the interest of single binocular vision or the dread of diplopia, as expressed by some, the right macula will be moved toward the image on the inner side of the retina. To accomplish this movement, the corneal pole of the eye will be moved outward to the temporal side. The eye is said to be abducted. The strongest prism which can be overcome when placed before the eye, base in, will be the measure of the abduction. If the patient can overcome a 7 diopter prism so placed, and an 8 diopter prism causes insuperable diplopia, the measure of the abducting power is 7 diopters. The same results will be obtained if the prisms are placed before either eye separately, or if the prism strength is divided between the two eyes. This fact will bear reiteration. While the one eye remains fixed, held by the stationary muscle light, the eye before which the prism is placed will be deviated; it represents the abducting power of both eyes. Here again the test is not one of muscle strength, but of a conjugate innervation, in which fusion plays an important role.

The average abduction varies from 4 to 8 diopters. In some instances it falls as low as 1 or 2 diopters, and in exceptional cases the abducting power may reach as high as 14 diopters. The latter is exceedingly rare. The average is nearer 4 than 8 diopters. In the same individual abduction is fairly constant from day to day. Like supraduction and infraduction, abduction does not admit of encouraging results in efforts to increase its strength by prism training. Within narrow limitations orthoptic exercises accomplish more than similar training in the vertical, but the results fall far short of what can be done by training the adducting power. The reasons for this are evident. Conjugate innervations for convergence call forth response almost every moment of conscious wakeful hours, and, therefore, it is in constant demand and training.

Divergence, on the other hand, occasionally spoken of as "negative convergence," is rarely called into action because of a total absence of our need for it, and the innervation is therefore a feebly developed innervation.

Adduction.—The ability to overcome prisms placed base out before either or both eyes is known as adduction. A distinction should be made between convergence and adduction. Both are innervational acts, and both depend upon a normal musculature to execute or to respond to the innervation. Convergence, however, is a normal physiological act which is unconsciously practised by every normal individual, whereas adduction is a drawing together of the visual axes against resistance, the prism furnishing the resistance in causing a separation of the images. The term "prism convergence" may be used synonymously with adduction.

To determine the maximum prism convergence, prisms are placed base out, before one eye in increasing strengths until the lateral displacement of the images cannot be increased without causing insuperable diplopia. The strongest prism which can be overcome without separation of the images is the measure of the adduction. Because of great possibilities in training by increasing strengths of prisms which can be overcome by an average patient, the measure of adduction may change from day to day in the same individual, and it becomes difficult to strike an average figure which might be called an average for all individuals. In fact, the adducting power, as given by various text-books, varies greatly. The discrepancy depends largely upon an individual operator's method of study. If the amount found at the first examination is selected, which is not a fair test for a patient, the average adducting power of all patients will probably not exceed 12 diopters. If, on the other hand, the highest prism diopters are taken as a final reading after repeated attempts are made, the results will also lack uniformity and will be unfair to the individual. A more or less concerted effort has been made to create a diplopia beyond the reach of fusion and record as a measure of adduction, the highest prism diopter which the patient is able to overcome by a gradual reduction in the strength of the prism. If this latter plan is followed the resulting figures will not represent the true fusion power of the patient. All figures will be too low. At best, the test is a difficult one for the average patient. It will be found necessary, therefore, in the taking of any duction to make two or three attempts before a correct reading is obtainable. If this same method is practised for each form of duction, the figures obtained will fairly represent *the duction* *power available for the individual in his daily needs.* Such measurements furnish a better working basis to account for the heterophoria present, and also point the way for proper treatment. A fair average of the strength of adduction, if this method is practised, is from 15 to 18 diopters.

Of vital importance from a clinical standpoint is the facility with which adduction can be trained in a majority of cases. After the patient has caught the knack of fusing prisms, 50 to 90 diopters may easily be overcome. This fact explains the reasons for the greater success from training in exophoria than in esophoria and hyperphoria.

The relation of abduction to adduction, when the measurements are taken at 6 meters, is of importance in orthophoria and in the lateral phorias. A ratio of 1 for abduction to 3 of adductions may be considered normal. In abnormal cases great variations are observed. Adduction is mostly in excess of abduction, but case records occasionally show an excess of abduction over that of adduction.

The amplitude of fusion horizontally is the sum total of adduction and abduction. This value, however, is not of so much importance clinically as the relation which one bears to the other.

Types of Prisms Used in Taking Ductions.—Measuring duction power by means of loose prisms placed in a trial frame is unsatisfactory, because the increase cannot be made smoothly and the tests are too time consuming. A prism battery, first suggested by Noyes, and modified by Gould (Fig. 50), is a slightly better method for taking lateral ductions but impracticable for vertical measurements. Gould's battery begins with 1 diopter prisms and increases 1 diopter at a step up to 20. The prisms are pivoted so that they may be placed bases out or in. This, too, is a rough test at best but a rapid method of arriving at approximate results. It possesses one advantage in that the prisms are equally divided between the two eyes.

A smoother method of increasing and diminishing the prism strength was devised by the late Dr. Samuel Risley and by Dr. Edward Jackson. Risley's rotating prism, the form which is now generally used, consists of two 15 diopter prisms placed in a frame with a rotating device, so that the prisms may be made to neutralize each other and may be rotated in opposite directions until a maximum of 30 diopters are obtained. The increase or decrease is made very gradually and smoothly. On the surface of the prism frame a scale is plainly marked, and one has but to read from the surface scale the degree of adduction or abduction as well as vertical duction (Fig. 51). The modern phorometers are equipped with

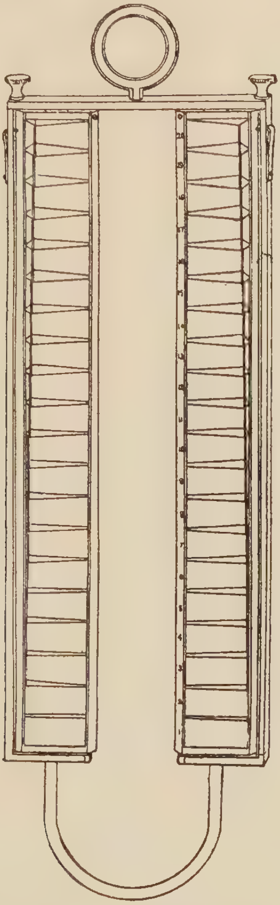

Fig. 50.—Gould's prism battery. (From "Ocular Muscles," by Hansell and Reber.)

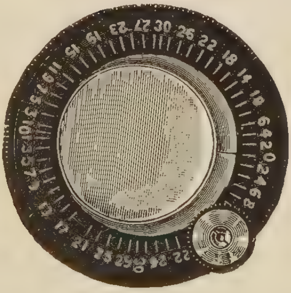

Fig. 51.—Risley's rotary prism. (From "Ocular Muscles," by Hansell and Reber.)

two Risley rotating prisms for use, one before each eye. There also is an advantage in being able to operate both prisms simultaneously. Chromatic aberration and prismatic astigmatism produced by the higher denominations of prisms tend to lower vision and, at the same time, tend to make the images before the eyes dissimilar—a condition which may interfere with fusion. It is desirable, therefore, to be able to divide the prisms before the eyes and in a measure overcome this difficulty.

Jackson's rotating prism consists of one 15 diopter stationary prism, around which are made to rotate two 7.5 diopter prisms. It is especially adapted to a study of the lower degrees of ductions because its operation is more delicate than the Risley prism.

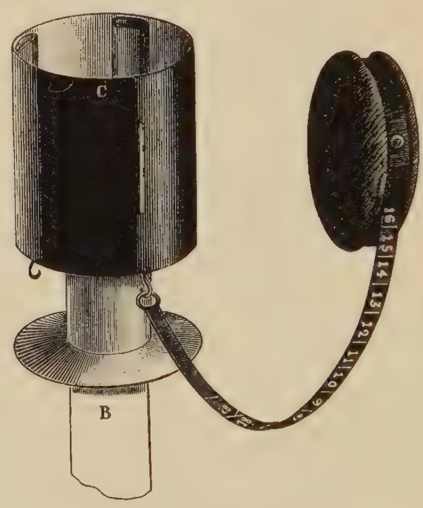

Fig. 52.—Landolt's ophthalmodynamometer.

Amplitude of Convergence.—A study of heterophoria would be incomplete without determining the convergence near-point and the amplitude of convergence. For the determination of the near-point of convergence, Landolt devised an ophthalmodynamometer, which is illustrated in Fig. 52.

It consists of a hollow black cylinder with a narrow vertical opening, back of which, within the cylinder, is placed a lamp. Attached to the cylinder is a tape, graduated in centimeters on one side and into the corresponding meter angles on the other. The near-point is determined by approximating the cylinder toward the patient, while the patient observes the illuminated slit. As the near-point is reached, the line tends to broaden, and as the slit passes inside the convergence near-point, the line becomes double. The distance of the slit from the eyes is measured by means of the tape in centimeters and in corresponding meter angles. The advantages of the method are, first, the ease with which the final approximate measure in centimeters and meter angles can be made; a second advantage lies in the fact that the illuminated line on the cylinder obviates the necessity of correcting for any hyperphoria which may be present. Its disadvantage is the necessity of multiplication of instruments when simpler methods are at hand.

Instead of an unnecessarily cumbersome instrument, such as the one described, a line placed on a card board will answer the purpose quite as well and is easily procured. Instead of a line, sometimes a dot is used for the same purpose. If any hyperphoria is present, however, measurements of the near-point may not be obtainable because of vertical diplopia. The line obviates this difficulty, as vertical displacement will not figure in the results.

For practical clinical work the author has found, as many others likewise, that a miniature electric light, such as is used in a luminous ophthalmoscope, serves as a subjective and an objective test at the same time. While the patient watches carefully for the appearance of two lights, the physician observes the position of the corneal images of the light in the pupillary areas. When the patient sees two lights the physician will see a change in the position of the corneal image of the light as one eye deviates outward. This point in convergence is carefully measured in centimeters. If there is a considerable degree of hyperphoria present it should be corrected before the near-point is taken. Care should be observed in the accurate measurement of this distance. According to Duane,[1] the meter stick should measure from a point 9.5 mm. from the apex when the eyes are directed straight forward, and to this measurement 25 mm. should be added, as the total distance of the convergence near-point from the interaxial base line. George, Toren and Lowell,[2] by means of an instrument, which they designated an "ocular kinemometer," found the center of rotation of the eyeball to be 15.4 mm. posterior to the corneal vertex and 1.65 mm. nasalward to the visual line. This fact, therefore, should be borne in mind in measuring the convergence near-point in order to obtain accurate results.

Given the convergence near-point in centimeters, its conversion into meter angles is accomplished by dividing 100 by the near-point expressed in centimeters. The quotient is meter angles of convergence. For example, a patient who can converge up to 12.5 cm. from the interaxial base line has 8 meter angles of convergence.

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[1] Arch. Ophthal., March, 1924.

[2] Am. Jour. Ophthal., October, 1923, p. 833.

In a discussion of the relation of the meter angle of convergence to the diopter of accommodation, the exact value of the meter angle was not given. This will depend upon the interaxial distance of the two eyes. We are apt to speak of this base line as the interpupillary base line, or as the base line which connects the centers of rotation of the eyeballs. Duane, very properly, in the communication in the *Archives of Ophthalmology*, quoted above, insists that this is incorrect. According to George, the center of rotation is not in the visual line, but 1.65 mm. to the nasal side of this line. A careful analysis of the chart suggested by Duane will convince the reader that the base line from which the convergence near-point is measured should be the intersection of the visual axes.

In order to convert meter angles into degrees, the exact measurement of the interaxial distance must be made. Duane advises the following method. In a dark room the exact position of the corneal reflex within the pupil is made of each eye separately, one eye being occluded, by directing the patient to look at the mirror which reflects the light into the eye, the operator taking his observation from behind the mirror. This, having been determined, the operator, facing the patient, closes his own right eye and occludes the patient's left. The meter measure is now so placed before the patient's eye that it will exactly mark the position of the visual line in the pupil, as previously determined, the patient fixing upon the operator's eye. The patient's right eye is now occluded and the operator's left is closed, the patient now fixing with his left eye on the physician's right. The position of the visual line is observed on the meter measure, under conditions similar to those observed in the right eye. This will yield fairly accurate results.

If, instead of the interpupillary distance, as suggested by Landolt, we substitute the term "interaxial distance" of a child of 58 mm., the value of the meter angle will be 1 degree 40 minutes, or 100 minutes. An interaxial base line of 64 mm. will make the meter angle 1 degree 50 minutes, or 110 minutes, in value. Nine-meter angles in the first instance would be equivalent to 15 degrees and in the second instance to 16.5 degrees. Ordinarily, however, the maximum convergence, sometimes called *positive convergence*, is expressed in linear measure, that is, the distance of the convergence near-point from the interaxial line, or it is expressed in meter angles.

The amplitude of convergence is the difference between the convergence near-point, also called positive convergence, and the maximum point of divergence, which is also called negative convergence.

Maximum divergence is measured by determining the prism abducting power of the patient obtained by placing prisms, base in, before one eye. Abduction varies, as has been shown from 4 to 8 prism diopters. A rapid, although only an approximately correct, way of converting the prism diopters thus obtained into meter angles is to divide the abduction by 7. If the abducting power obtained is 7 diopters, 7 divided by 7 would equal 1 meter angle of maximum divergence or minimum negative convergence. If abduction is 8 prism diopters, 8 divided by 7 would be the number of meter angles, or 1.1 meter angles of maximum divergence. This method is sufficiently accurate and rapid to make the more correct method of calculation unnecessary.

For those who wish to follow the calculation through, the following method is given: In an interaxial distance of 64 mm. the value of the meter angle is 1 degree 50 minutes, or 110 minutes, or its equivalent in fraction form, 110/60 degree. In the above example of 7 diopters of divergence the value of each prism diopter is 0.57295 degrees. The value of 7 diopters is 7 times 0.57295 degrees or 4.01 degrees. If each meter angle has a value of 110/60 degrees, 110/60 degrees divided into 4.01 degrees equals 2.19 meter angles. This, however, represents the total divergence of the two eyes as the one eye remains fixed. In the diverging act, but one-half the amount will be required from each eye. The meter angle of divergence of each eye will be one-half of 2.19, or 1.09 meter angles.

The amplitude or range of convergence, therefore, is the maximum divergence plus the maximum convergence. In orthophoria, or a normal muscle balance, Landolt believes that 9 meter angles of convergence are necessary for comfortable sustained close work. This will allow 3 meter angles of convergence required for the average normal distance of close work, 33 cm. Six-meter angles will be held in reserve. A reserve of less than 6 meter angles in Landolt's judgment will be insufficient. In this opinion most authorities concur. The reader will appreciate, therefore, the importance of carefully determining the convergence near-point and the corresponding meter angle of convergence in all cases of lateral heterophoria.

Landolt's improved model of double prisms made by Zeiss, graduated in meter angles and diopters, facilitates the measuring of the angle of divergence, but the ready method given above, as suggested by Hansell and Reber is equally simple and practical.

METHODS OF DETERMINING THE MUSCLE BALANCE.

The various tests employed in determining the muscle balance have as their underlying principle the changing of the retinal image of one eye as to color, size, shape, or position, so that fusion will not operate in the interests of single binocular vision, and a latent tendency to heterophoria will become manifest, the degree and character of which can be carefully determined.

The tests may be arranged in three groups: (1) Displacement, or diplopia tests; (2) distortion tests; (3) cover tests.

Displacement, or Diplopia Tests. The displacement test, which was suggested by von Graefe, consists in creating insuperable diplopia in a vertical direction to determine the presence of a lateral imbalance, and insuperable diplopia in a lateral direction to make manifest a vertical deviation.

If an 8 diopter prism is placed base down before the right eye the right image will be displaced upward so that the two images cannot be fused in an average case. If the fusion faculty is capable of bringing the two images together a prism of a higher denomination must be employed. This will rarely be found to be necessary. With the images displaced in a vertical direction, the operator determines from the patient's answers whether the two images are in a vertical line. If they are not, the next point to determine is, to which side of the lower light the upper image is located. If the upper image, the image of the right eye, is situated to the right, honomymous displacement, the condition is one of esophoria. As a prism displaces the image toward the apex, the strength of prism, placed base out, which will bring the upper image directly in a vertical line with the lower image, will be the measure of the esophoria in prism diopters or centrads. If the upper image is found to be to the left of the lower light, the condition will be a crossed displacement, or exophoria. The strength of prism, placed in the frame, base in, before either eye, which will bring the images, one over the other in a vertical direction, is the measure of the exophoria in prism diopters. These measurements are correct only when the light is placed at a distance of 6 meters from the patient.

To detect and to measure a vertical deviation, a displacing prism of 8 diopters is placed in the trial frame, base in, before either eye. If a prism of 8 diopters is insufficient to produce insuperable diplopia in a lateral direction, a prism of a higher denomination must be employed. Because of the greater strength of the converging power of the eyes, and the ability of the patient to overcome prisms of high refracting power, the prism is not placed base out. If placed before the right eye the right image will be displaced to the right. If the right image is in the same horizontal plane as the left, there is no tendency to latent deviation in the vertical, and the condition is one of orthophoria in the vertical direction. If the right image is above the left the condition is left hyperphoria. That is, the left eye is higher than the right and the left image, therefore, is lower than the right. The strength of prism, placed base down before the hyperphoric eye, which will restore the images to a common level, is the measure of the left hyperphoria in prism diopters. If the right image is below the left, the condition is right hyperphoria. The prism placed base down before the right eye, which will raise the right image to the level of the left, is the measure of the degree of right hyperphoria in prism diopters.

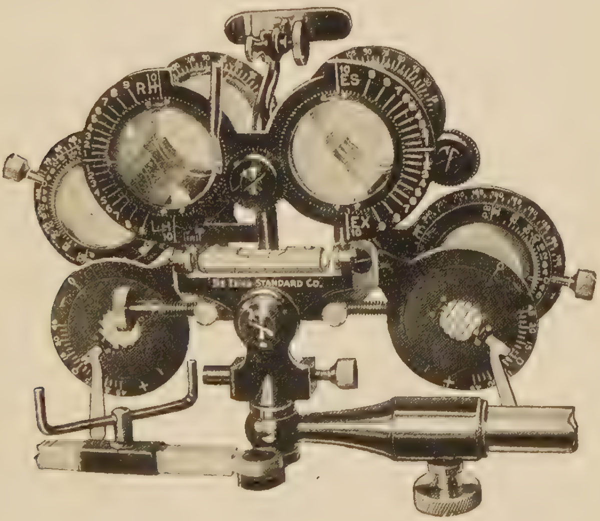

Fig. 53.—Stevens' phorometer.

In Stevens' phorometer two 5 diopter prisms are so placed in revolving frames, controlled by a lever, that the prism strengths are divided between the eyes, and they can correct displacement either in a vertical or a horizontal direction.[1] By a simple rotation of the handle the images can be brought into alignment, either in the vertical or the horizontal, and the degree of the phoria and the character of the same is read directly from the face of the prism frames, which are accurately graduated (Fig. 53).

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[1] Artificial vertical or horizontal diplopia is induced, as in the von Graefe test, by a loose prism placed back of the rotating prisms in an appropriate cell.

Although the method of testing the balance on the phorometer is simplified and quick of execution, the beginner will do well to practise at first with loose prisms in order to become familiar with the technic and the principles involved. In placing loose prisms in the trial frames, great care must be observed that the prisms are accurately placed, lest an artificial phoria be created in the direction in which the measurements are being taken.

In taking the muscle balance, either by this method or by any other method, the reader is reminded that he is not measuring innervations, nor is he measuring the strength of an individual muscle. The fusion faculty is in abeyance and should not be operative. The position which one eye assumes in relation to the other is not the result of over- or under-action of a single muscle, but of the combined action of all the muscles while the eyes stand "attention."

Maddox Double Prism.—Maddox arranged two 4 diopter prisms in a cell suitable for a trial frame, as shown in Figs. 54 and 55. The prisms are placed with bases together, so that the image before which the prism is placed will be converted into two—one above and the other below the horizontal. The image of the other eye will appear midway between. If the middle image is to either side of the vertical line, which would connect the double images, the patient has esophoria or exophoria and the prism placed base in or out before either eye, which will bring the three images into alignment, will be the measure of the esophoria or exophoria. In order to measure hyperphoria the double prism should be placed horizontally. In this position, however, the results are not so satisfactory because the displacement of the images, as a rule, is not sufficient to entirely eliminate the fusion influence. Even in determining lateral imbalance, the many images which the patient must consider is confusing and the results are difficult to obtain. It is at times a difficult matter to obtain satisfactory answers from patients, and in taking a muscle balance the least complicated method which is employed usually proves to be the most satisfactory.

Instead of the Maddox double prism, Thorington's truncated prism, made of red glass or cobalt blue, may be substituted. In this prism the two prismatic bases are separated by a breadth of

PLATE III

FIG. 1

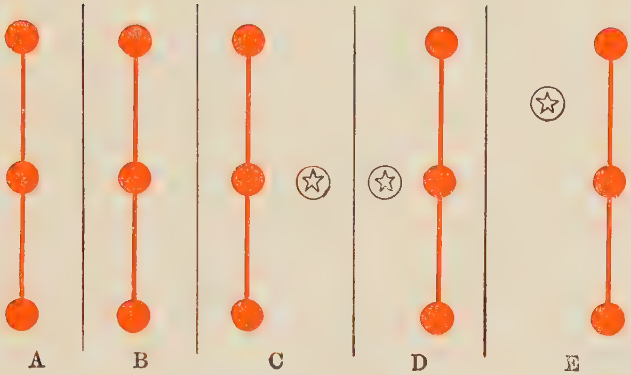
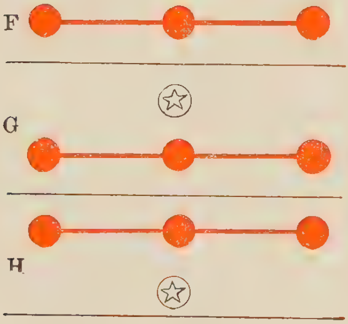


FIG. 2



The Effect of Thorington's Double Prism of Ruby Red.

FIG. 1.—*A*, three images connected by streak; *B*, no lateral deviation; *C*, exophoria; *D*, exophoria; *E*, left hyperexophoria. (From Thorington's "Prisms.")

FIG. 2.—*F*, no vertical deviation; *G*, left hyperphoria; *H*, right hyperphoria. (From Thorington's "Prisms.")

plain glass. Either the red or the cobalt blue truncated prism is better than the Maddox prism if a test of this character is desired.

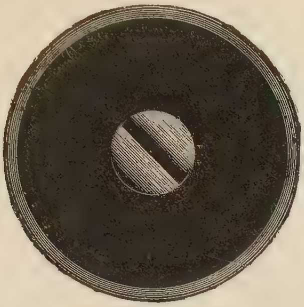


Fig. 54.—Maddox double prism. (From "Ocular Muscles," by Hansell and Reber.)

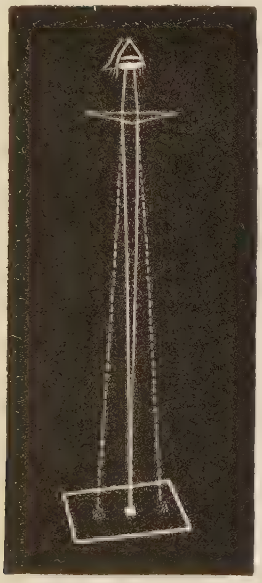
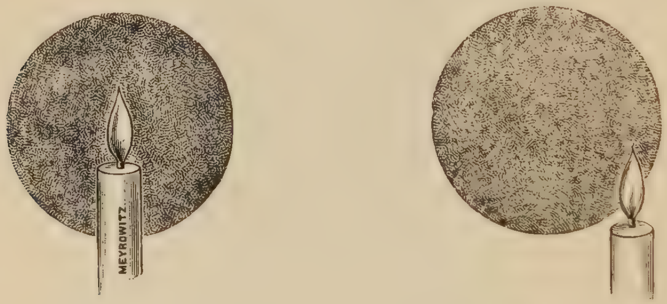


Fig. 55.—Effect of a double prism on a beam of light (Maddox.) (From "Ocular Muscles," by Hansell and Reber.)

Distorting Tests.—These tests depend upon the distortion of one image in order to suppress or to remove the desire for fusion. Three are to be considered, the cobalt-blue glass, the convex sphere and the Maddox red.

Cobalt-blue Glass.—A glass stained with cobalt oxide possesses the power of intercepting all the rays of the spectrum, excepting the red and blue. When such a glass is placed before an emmetropic eye the image will be much smaller than the real image, and the center of the image will be blue surrounded by a red halo. The same arrangement of colors will be present in hypermetropia, excepting that the red will not be arranged in a circular manner if astigmatism is present. In myopia the center of the image will be red and the halo will be blue. Hansell and Reber believe that this test has some advantage over other tests, in that the cobalt glass does not refract the rays of light entering the eye, in front of which it is placed. They believe that any deviation which the test reveals or uncovers is true muscular insufficiency. The test can only be used when there is an appreciable amount of heterophoria present because in low degrees there will be a tendency to fuse even though one image is distorted and discolored. In fact, the author believes that the test is only practicable in hyperphoria of 2 or more diopters. In the lateral a true measure is difficult because of the desire to fuse unless the lateral tendency is of considerable degree. It is a test which is not often employed in routine practice.



Orthophoria. Heterophoria.

Fig. 56.—The convex spherical test. (From "Ocular Muscles," by Hansell and Reber.)

The Convex Sphere.—If, instead of a cobalt-blue glass, a convex sphere of 20 diopters is placed in a trial frame before one eye and a pin-hole disk is placed back of the lens so as to cut off all of the sphere but the center, the muscle light will have the appearance of a luminous disk of light, as illustrated in Fig. 56. If orthophoria prevails the true image before the other eye will consist of a bright dot or flame in the center of the luminous disk of light. If heterophoria is present the true image will not be in the center of the disk, but will be displaced laterally or vertically. The prism or prisms placed laterally and vertically, which will make the true image to occupy the center of the disk will be the measure of the heterophoria.

This test also has a serious objection. The sphere must be most accurately centered in the trial frame before the patient's eye or an artificial heterophoria will be created. It is most difficult to correctly place the sphere to obviate this error and for this reason the author has not found the test of much value.

The Maddox Rod.—The Maddox rod consists virtually of a glass rod mounted on a black disk in lens form, and fitted to a trial frame (Fig. 57). The principle of the rod lies in the fact that a rod so mounted refracts in only one direction, that at right angles to its axis. It therefore converts a luminous point of light into a line of light. When a Maddox rod is placed before one eye, a muscle light at 6 meters distance will look like a line of light. If the patient has orthophoria, the true image of the muscle light will be found directly in the line of light or the line of light will be said to pass through the light. If the rod is placed before the right eye at axis 90, the line will appear in the horizontal meridian. In right hyperphoria the line will be below the light and the prism placed base down before the right eye which will bring the line up to the level of the light is the measure of the hyperphoria in prism diopters. If the line is above the light, the condition will be left hyperphoria. To explain the reason for this displacement in hyperphoria, whether right or left, the reader should recall that in right hyperphoria the right eye is higher than the left, and its image must, therefore, be below, according to the laws of projection and direction.

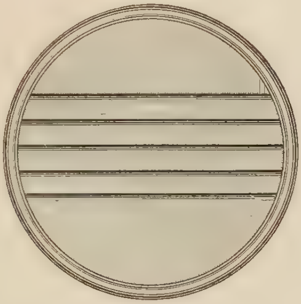


Fig. 57.—Maddox double rod. (From Thorington's "Prisms.")

To measure esophoria and exophoria, the axis of the rod should be placed at 180 degrees in the trial frame. The line of light will then appear in a vertical direction. If the rod is before the right eye and it is seen by the patient to the right of the light, the condition is one of esophoria, homonymous displacement. The degree of esophoria will be determined by the prism placed base out which will cause the line to pass through the light. If the line is to the patient's left, *i. e.*, crossed displacement, the condition is exophoria. The amount of exophoria is measured by prisms placed base in before either eye, which will cause the line to pass through the light.

This test is the most practical, of easy application, and the one usually employed in every-day practice. Steven's phorometer is equipped with two Maddox rods so that the test can be varied by placing it before either eye. In addition, the double rotating prisms attached, quickly record the amount of the heterophoria which may be read from the face of the prisms (Fig. 53).

Instead of a single rod, Maddox also devised a compound rod by placing three or four rods together. This has the advantage of giving better definition to the line of light. It is the author's experience, however, that few of the compound rods found on the market are strictly accurate. When a compound rod is purchased, it should be tested carefully at 6 meters before one eye and its position in relation to the light should be noted. It should then be reversed before the *same eye* to note whether the line occupies exactly the same place as in the first test. If the rod is perfectly made, the results should be the same.

The author finds it difficult for the average patient to see the line at the first test. A red line is more easily picked up than a line of the same color as the light. A red rod, therefore, may be found to be better, although a red line as a rule is not so brilliant as a yellow line. When a patient experiences difficulty in noting the presence of the line, the operator can aid greatly by alternately covering and uncovering the exposed eye or by slightly rotating the rod so as to cause the line of light to move. Much patience must be exercised in some individuals in order to make the test satisfactory.

We are concerned at this time with the technic and the principles of the tests. It is pertinent to observe, however, (1) that a careful muscle balance should always be taken as a routine in the examination of each patient at the first visit when this is possible; (2) that a study of the muscle balance should not be made when the eyes are under the influence of a cycloplegic; (3) that a final study of the muscle balance should be made *with the patient's proposed correction on* before the final prescription is written for the patient's glasses. Unless these rules are followed, the physician's work in muscle testing may fall into error, and the true value of the careful consideration of a patient's muscle balance will not be fully appreciated.

Tests for the Reading Distance.—One can more fully appreciate the discomfort and the symptoms which may follow considerable degrees of muscle imbalance laterally, and of even low degrees of displacement in the vertical, when it is recalled that 1 diopter of disturbance will mean a displacement of 1 cm. for each meter's distance. At 6 meters 1 diopter of hyperphoria will mean a displacement of 6 cm.; 2 diopters will cause a displacement of 12 cm., etc. This must be overcome by the fusion faculty and the effort entailed must of necessity give rise to fatigue and a train of reflex nervous symptoms. At the working distance, however, even more careful coördination is required and the attending symptoms are more far-reaching and disturbing. This is especially true of vertical disturbance. It becomes a necessary corollary of muscle balance studies at infinity, *that the near-point study is equally important.*

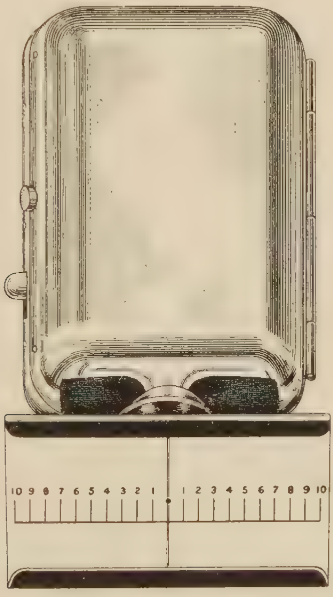


FIG. 58.—Thorington muscle light.

The Maddox Rod Test for the Near-point.—We observed that the Maddox rod test for distance was most practical and satisfactory. It is equally true for the near-point. Thorington has contributed a muscle light test to be used with the Maddox rod for the near-point, which is of great value and time-saving. A small dry-cell lamp is placed back of a pin-point opening in the center of a white flat surface of celluloid, upon which is marked a scale of 10 diopters to each side of the light. The amount of displacement of the line from the Maddox rod is read off in diopters and obviates the necessity of using prisms to measure the degree of the heterophoria. It is fairly accurate when used at a distance of exactly 33 cm. (Fig. 58). If the Thorington muscle light is not available, the small light in a luminous ophthalmoscope may be used. The light is held exactly 33 cm. in front of the patient, and prisms may be placed in the trial frame, bases in or out, up or down, until the line passes through the light.

Maddox Wing Test for the Near-point.—In the so-called wing test of Maddox, the scale for hyperphoria marked in red figures, and the white scale for lateral disturbance are seen by the left eye. The lower right-hand quadrant of the wing is only visible to the right eye. In this quadrant is placed a horizontal red line with an arrow head which points to the red scale. Without difficulty the patient can determine toward which figure in the red scale the arrow points. The reading of the right or left hyperphoria is direct and expressed in prism diopters on the scale.

In the same quadrant (the lower right) the right eye sees a hand with the index finger pointing to a carefully defined white scale of figures. The figure to which the finger points is the measure of the esophoria or exophoria in prism diopters.

This test to most people is simple and the muscle balance in the horizontal and vertical is quickly determined. Some, however, find great difficulty in seeing the entire surface simultaneously, and in a number of instances, the author found it necessary to abandon the test. On the whole, however, the wing test is most satisfactory and is time-saving. It probably is more accurate in determining latent heterophoria than any other test for the near-point. In the test, the eyes are completely disassociated, and the images before the two eyes are totally different.

Cover Tests.—The cover tests furnish a ready means of detecting the presence of heterophoria. The objective cover test is applied as follows: With both eyes uncovered, the patient is instructed to fix on a candle or muscle light 6 meters distant. One eye is now occluded by a convenient cover. As the cover is rapidly shifted from one eye to the other by the physician, he carefully notes whether the eyes remain fixed or move under the test. If they do not move, the muscle balance is probably normal, or the amount of heterophoria, if present, is of very slight degree. If the eye which is under cover moves in the same direction as the cover is carried to the opposite eye, the condition is one of exophoria. While the eye is under cover it moves out to the extent of the latent squint present and in order to fix, when it in turn is uncovered, it must of necessity move in. The eyes are, therefore, said to be "out under cover." If the covered eye moves in a direction opposite to the movement of the cover, as the covered eye is now exposed, the condition is esophoria. In this instance, the eyes are said to be "in under cover."

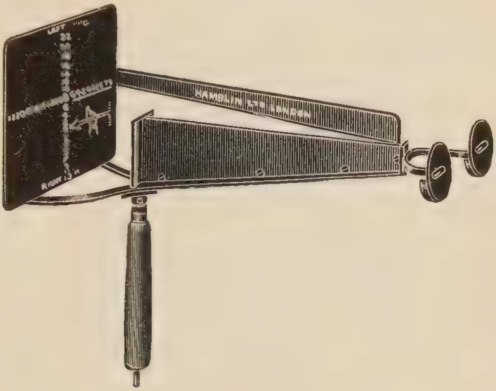


FIG. 59.—Maddox wing test.

When hyperphoria is present, the covered eye will move up or down as the cover is shifted. If it moves up, the eye is lower in position than the fellow eye, and if it moves down, it is higher than the fellow eye. As the cover is reversed, the movement of the fellow eye will be directly opposite. If the right eye, for example, moves down as it is uncovered, the left eye will move up as it in turn is uncovered. The eye which is high is called the hyperphoric eye. In the example, the condition is right hyperphoria.

This cover test may also be made a subjective test which Duane calls "parallax test." If the eye under cover wanders because of a latent deviation, as it becomes the fixing eye, the patient will be conscious of a sudden movement of the image. The movement will be opposite to the position which the eye assumed under cover. In exophoria, the movement will be in the direction in which the cover moves. In esophoria, the movement will be opposite to the cover.

In both tests corrective prisms may be applied to stop the objective and subjective movement. While the parallax test is not used as a routine examination, the objective test is quickly performed and is of great value in detecting the presence of hyperphoria.

HYPERPHORIA.

Definition.—By hyperphoria is meant a tendency to a latent deviation of one visual axis above the plane of the other. It differs from hypertropia in that it is latent instead of manifest. In most instances it can be overcome by an effort on the part of the patient, but as a rule at the expense of symptoms to be described later. In hypertropia, the displaced image is so far beyond the reach of the fusion faculty, that no effort is made to correct the deviation, and because the false image is suppressed, no symptom of discomfort, other than the squint, becomes apparent.

The terms right and left are employed to indicate which visual axis is the higher. It should be noted that the visual axis, and not the position of the image, is used to designate the character of the hyperphoria. The eye which is the higher is called the hyperphoric eye and not the one whose image is high. This thought should be borne in mind in the correction of hyperphoria by means of prisms. The correcting prism displaces the image toward its apex and is, therefore, placed base down before the hyperphoric eye, in order to raise the image to the level of the other. The terms right and left do not indicate whether the hyperphoric eye is too high or the hypophoric eye is too low. It simply indicates the eye which is the higher.

General Considerations.—In a study of specific types of heterophoria, it is important for the student to realize just why any form of latent deviation, and particularly hyperphoria, should be so serious a factor in the development of muscular asthenopia. The chief source of trouble is found in the degree of separation of the two images. At 6 meters a 1 diopter displacement is equivalent to 6 cm. in tangential measurement; 2 diopters equals 12 cm., etc. In other words, a patient who has 1 diopter of hyperphoria when the fusion faculty is held in abeyance, will show a separation of 6 cm. between the two images at a distance of 6 meters. If he has 4 diopters the separation of the images will amount to 24 cm. In the lateral forms of heterophoria, esophoria and exophoria, high degrees of separation of the images is not so important as in vertical displacements. In esophoria it is of considerable importance, but in hyperphoria it is paramount.

Two facts should be recalled to fully appreciate this. First, in the development of binocular vision, stress was laid upon the presence of a good vertical balance of the eyes at the time of birth. Vertical balance is well established in the average normal new-born. This, in itself, may not seem to be a fact of great importance. When, however, the second fact is considered, namely the low degrees of vertical deviations which can be overcome, as shown in infraduction and supraduction, the first fact gains in importance. The average supraduction is 3 prism diopters and the same is true of infraduction. Few individuals can overcome a separation in the vertical of more than 3 prism diopters. Beyond this an insuperable diplopia may be produced.

Inability of the average patient to overcome any great vertical displacement of the images accounts for two well-known clinical observations: (1) Failure of prism training as a corrective measure in hyperphoria; (2) the great suffering of patients with even moderately low degrees of hyperphoria, and the great inconvenience of vertical diplopia in paralytic squint as compared with lateral deviations. Hyperphoria, therefore, is perhaps the most important form of heterophoria to be considered. Fortunately, the possibility of correction by means of prisms is one of its encouraging features.

Frequency of Hyperphoria.—Statistics, as to the frequency of hyperphoria must be drawn largely from private case records because routine muscle studied are rarely made in hospitals. Hansell and Reber, in 700 consecutive case studies, found 20 per cent in which there was 0.5 degree or more of hyperphoria. In 3600 cases, they found 7 per cent who had 1 degree or more. Carpenter reported 35 per cent; Posey, 13 per cent; Bannister, 7 per cent; Howe and Williams, 16 per cent. My own records in a series of about 800 cases approximate those of Carpenter, namely about 33 per cent. If the test is made at the near-point, as it should be, as well as at 6 meters, the percentage increases. A factor which may influence statistics to an appreciable extent, is the study of the muscle balance with the patient's ametropic correction worn. It is not unusual to see lateral, as well as vertical deviations, diappear with the wearing of a proper correction. The relation of ametropia to muscle imbalance is quite intimate. The cause and effect relation of these two conditions in certain cases, and the persistance of hyperphoria, in spite of the ametropic correction in another group or cases, warrant the assertion that no refraction is complete without a careful muscle study for distance and the near-point.

Etiology.—In a study of the underlying factors, a point which needs emphasis, is the influence of age. Hansell and Reber found 33 per cent occurred in patients under thirty years of age, and 66 per cent in patients over thirty years. This is in accord with the author's experience. Muscle weakness or muscle over-action, abnormalities in attachments and insertions, and orbital changes, have all been assigned as causes. In youth a strong fusion faculty, and a superabundance of elasticity render perfect coördination easy. Incoördination is readily held in check. With maturity in years, latent tendencies become more manifest. In addition to this natural development of an existing but quiescent condition, adult life contributes its own factors. Muscle fatigue is more common in adult life than in youth. Occupational influence, long hours of sustained close work, the gradual changes in muscle structure incident to age, from which the ocular muscles can not claim exemption, and the tension of modern methods of life are all factors which are contributed by advancing years.

In addition to these causes, which can account for a large number of the constant variety of hyperphoria, namely those which do not show much variation from day to day, neurotic influences are often responsible for a type of heterophoria which shows great variation. It may be this influence which increases or aggravates a vertical deviation feebly present from early life, or by it, a vertical deviation may be brought into existence which is characterized by great variation in degree. The latter is a special form of imbalance which requires a form of treatment differing radically from the usual methods of treating heterophoria.

The relation of hyperphoria to lateral disturbance, esophoria and exophoria, is interesting. As mentioned above, moderate degrees of lateral imbalance and vertical disturbance are corrected simultaneously with the correction of any existing ametropia. Other cases continue to show a residual disturbance in the vertical and laterally even after a proper correction is worn. It is a common observation that lateral disturbances often disappear after the correction of the hyperphoria by prisms placed in the vertical. This is more frequently the rule than the opposite condition. In other words, lateral deviations are more frequently caused or seem to be caused by hyperphoria than is hyperphoria dependent upon lateral disturbance. One cannot ignore the fact, however, that both forms of heterophoria are intimately bound together or associated in the same functional disturbance and that the correction of one is often followed by a correction of the other.

Symptoms.—The symptom complex of muscular asthenopia, like that of ametropia, is varied. In fact so similar are these two types of asthenopia that it becomes difficult at times to differentiate between them. In not a few cases, the symptoms are the result of the combined action of both causes. If, however, the ametropia is corrected and asthenopic symptoms continue, the cause at once becomes clear. In a given number of cases, the relief of the ametropia by suitable glasses is followed by the entire relief of all symptoms. When this occurs, not a few oculists advise against the correction of the hyperphoria because the patient experiences no immediate symptoms which are regarded as of muscular origin. Many of these patients return, however, with their former symptoms, and the proper incorporation of prisms in the glasses to be worn, usually results in entire relief.

Headache. - In the enumeration of symptoms of which the patient complains, headache leads in frequency. The pains are supraorbital, temporal and even in the eyeballs themselves. Less frequently, they may be occipital and occasionally on the top of the head. They are aggravated by crowds, by riding in a train, a trolley, or an automobile and by watching games intently, such as baseball, especially when the patient becomes tense from great interest in the game. The moving picture screen is a commonly observed cause of this type of headache. They may also be described as "shopping headaches." The term *panoramic headache* is a most suitable phrase to use in characterizing, from an etiological standpoint, the type of headache under discussion. While the "panoramic headache" is also observed in pure ametropia, the term is best reserved for the muscular type, as seen in hyperphoria. The headache as a rule is increased by close work, and usually gathers force as the day advances. A night's rest brings relief.

Drowsiness on Close Work.—Many patients are fortunately exempt from headaches, but suffer from other forms of asthenopia. Drowsiness upon the slightest attempt at doing close work is almost as frequently observed as headache. Hyperphoria is not the only muscular condition which can account for this symptom, as it is noted in any form of heterophoria. It is also encountered in pure forms of refractive errors and in uncorrected presbyopia. It is, nevertheless, a common symptom in hyperphoria.

Lid Disturbances.—Chronic hyperemia of the lids, marginal blepharitis, and blepharospasm are frequently observed in hyperphoria as well as in other types of muscular asthenopia. A correction of the hyperphoria is followed by prompt relief.

Defective Binocular Vision.—During the testing for refractive errors, inability of the patient to read as well or as correctly with both eyes as with each eye separately, is an indication that hyperphoria of a considerable degree is present. If one recalls the fact that an average patient can overcome but a moderate amount of displacement of the images vertically, and if one appreciates the fact that every degree of hyperphoria means a separation of the images 6 cm. at 6 meters, the reasons for defective binocular vision are easily understood. It is of greater annoyance to the patient than the defective vision due to refractive errors. In fact, the latter form of defective vision is rarely noted by the patient unless the defect is very great. Lowered vision from ametropia is so rarely accompanied by asthenopia, that it often is the cause for the failure of many patients to consult an oculist when they are urgently in need of his services. The annoying blur from hyperphoria, on the other hand, is often the cause of the patient's return to the oculist with the complaint that his "glasses are not satisfactory."

Facial Expression.—Among the symptoms thus far described, panoramic headache and defective vision of the peculiar variety just discussed may be called characteristic of hyperphoria. Little attention has been given to the changed expression in the face of a patient suffering from hyperphoria, although it is of common occurrence. The arched or furrowed brow is not characteristic of a vertical muscular disturbance, but it, together with drawn features, are sufficient to indicate that such a patient is suffering from some form of discomfort.

Head Carriage.—More characteristic is the tendency for the patient to pose as it were with the head tipped to the right or left shoulder. "Shoulder tipping" is observed in muscle palsies along with turning of the face up or down, right or left. A careful study of a series of patients accustomed to tip the head to either shoulder, reveals the fact that most of them are victims of uncorrected hyperphoria. The head is tipped, as a rule, to the shoulder on the side of the hyperphoric eye. It is a mechanical effort on the part of the patient to bring the images of the two eyes to a common level. It differs from true vicarious rotations of the head found in muscle palsies, although occasionally it is found in this latter condition with vicarious rotations. (For a more detailed differentiation between the two conditions, the reader is referred to the Section on Organic Palsies or Paralytic Squint.)

Diagnosis.—In the diagnosis of hyperphoria, we are concerned not only with its detection, but also with an accurate estimation of the amount or degree of the separation of the images. While one may, therefore, employ one of the several methods at our disposal for its detection, the measurements should be made by methods which are known to be accurate.

Objective Cover Test.—The cover test, objectively applied, furnishes a fairly accurate means for its detection. The patient is asked to look at a small object, preferably a small light at 6 meters distance, while the cover is passed from one eye to the other. If there is an appreciable amount of hyperphoria present, the eye under cover will be observed to move upward or downward as the case may be. The eye which moves downward, as the cover is passed to the fellow eye, is the hyperphoric eye. The eye which moves upward as it is uncovered, is the low eye or hypophoric eye. Thus, if the right eye is under cover, it will move up or down as the cover is suddenly passed to the left eye, if hyperphoria is present. A downward movement will indicate the presence of right hyperphoria; if it moves upward, the condition is one of left hyperphoria.

The test may be made most accurate for the detection of even low degrees of hyperphoria, if the light from a luminous ophthalmoscope be thrown upon the cornea and note is made of the position of the corneal reflex with reference to the pupillary border.

Parallax Test of Duane, or the Subjective Cover Test.—The value of the test just given may be supplemented by Duane's parallax test. When the cover is suddenly passed from one eye to the other, if hyperphoria is present, the patient will observe that the luminous point upon which he fixes will tend to move up or down. The movement will be opposite to the position of the eye, as it is covered. For example, if the right eye under cover deviates upward in the position of right hyperphoria, the luminous point will move downward as the cover is passed to the left eye. Duane calls the condition "right parallax." If the eye is low as in left hyperphoria, the movement will be upward as the cover is passed to the left eye, "left parallax."

The prism placed base down before the hyperphoric eye, which will abolish the objective and subjective movements in these two tests is the measure of the hyperphoria. While these tests are of great value, in the detection of hyperphoria and rapidly executed, the measuring of the degree of deviation, by the placing of prisms to overcome the movements, is rarely practised because of more rapid methods at our disposal.

Prism Test of von Graefe.—In this test, an artificial lateral diplopia is created by placing a prism of 8, or if necessary 10 diopters, base in before one eye. If hyperphoria is present, the two images will not be on the same level. The low image belongs to the hyperphoric eye. A prism placed base down before the eye of low image, which will raise it to the level of the other image, will be the measure of the hyperphoria.

This test has been somewhat simplified on all phorometers, by a device popularized by Stevens. In this device, illustrated in Fig. 53, two prisms, each of 5 diopter strength, are so arranged that they can be rotated into a vertical or horizontal position. Lateral displacement is obtained by an 8 diopter prism placed base in before one eye. By proper rotation, the hyperphoria is corrected and the degree of the hyperphoria is read off from the scale on the front of the device. Other rotary prisms have been employed from time to time in a similar manner, but the Stevens phorometer is probably the best type now in use.

Maddox has very correctly called attention to the fact that in any test devised for the detection of hyperphoria by means of prisms, the desire for fusion is not wholly removed, but the power to fuse is taken away. If, therefore, the images are not sufficiently separated, there may be some error in the estimation of the degree of hyperphoria. If the separation of the images is sufficient to remove all desire for fusion, the test is fairly accurate.

Although the tests just described have fallen more or less into disuse because of the ease with which the next test to be described is practised, the student will profit by practising them. They tend to cultivate the habit of accurate observation and familiarize one with the effect and the technic of prism use. They also help greatly in assisting the student to visualize the corrective measures necessary to overcome latent forms of deviation.

Maddox Rod Test.—This test, described in a former section, is probably used more than any other. If the rod is true, it is quite accurate for routine work. Each rod should be tried out by the physician before it is placed in his trial case for use, by making careful observations on his own eyes. The rod is placed horizontally and its position with reference to the light should be observed. It should then be rotated 180 degrees in the trial frame, and the line should again be noted. If the result is exactly the same in each test, it may be regarded as accurate.

The double Maddox rod of red glass is probably the best for general use. Many patients experience difficulty in recognizing the line, and the compound rod renders the line more pronounced. The rod may be placed before either eye, but a routine habit of placing the rod regularly before the right or the left, as the physician may elect, will avoid mistakes and will require less concentration on his part during the examination. It is the author's habit to place the rod before the left eye unless the vision in the left eye is considerably lowered. In the event of lowered central vision, it is best to place the rod before the eye of greatest visual acuity. When placed before the left eye, if the patient sees the rod above the light, it is right hyperphoria; if the line is below the light, the condition is left hyperphoria. The prism placed base down before the hyperphoric eye which will cause the line to pass through the light, is the measure of the hyperphoria.

The same test is one of the best for measuring hyperphoria at the near-point. A small electric light, such as is found in a luminous ophthalmoscope, is held in front of the patient at a distance of 33 cm. or 13 inches, and the examination is made as it was for distance. For the application of this test at 6 meters or at 33 cm., the Stevens phorometer is satisfactory and time-saving. The Maddox rod is rapidly moved into position, and as the double prism arrangement in front of the Maddox rod quickly raises or lowers the rod, the exact measurement of the hyperphoria is read from the scale which appears on the face of the frame which holds the prisms in place.

Instead of using a small electric light, the Thorington muscle test may be substituted. This consists of a pin-point of light, which appears in the center of a carefully marked scale, each mark on the scale representing 1 degree of displacement up to 10. If the light is held accurately at a distance of 33 cm., the scale is fairly accurate and considerable time is saved in making the test.

For the measurement of vertical disturbance at the near-point, the Maddox wing test is to be recommended. This instrument is so constructed that the left eye sees the vertical scale in red figures, and the horizontal scale in white figures. The right eye observes the red pointer, corresponding to the Maddox rod, for hyperphoria and the white pointer for the lateral balance or imbalance. In testing hyperphoria, the patient notes carefully to which figure in the vertical red scale the red line points (or through which figure the red line passes), the figure indicating the degrees in prism diopters of the right or left hyperphoria as the case may be. For the taking of the muscle balance at the near-point this wing test, rapid in execution and easy for the patient, is quite as accurate as any test which the author has employed. Although the multiplication of instruments is not always desirable, the busy man will find its purchase worth while.

Many other tests of merit have been in use, such as the Prentice cross, Carpenter's dot test, and a host of others, but in each instance the same general objection applies, namely the purchase of another instrument. If a test does not add materially in shortening the time required in the examination of a patient, or in furnishing more accurate results than the practical tests now in use, it cannot be regarded as an essential to one's equipment.

Prolonged Occlusion of One Eye.—When no great amount of hyperphoria becomes manifest by the methods advised and asthenopic symptoms continue, or in those cases in which the amount of hyperphoria varies at different examinations, the occlusion test should be practised. This test consists in the occlusion of one eye for variable periods by means of a bandage, and measurements for heterophoria are made after the bandage is removed. To be of real value, the occlusion should be practised over a period of days and not hours, as is the usual custom. When the bandage is kept in place sufficiently long, the occluded eye is apt to fall into its natural position of physiological rest. As full conscious effort to obtain binocular vision does not restore the two eyes to a normal level immediately after uncovering the occluded eye, a considerable degree of hyperphoria may be uncovered. These results are not obtainable after occlusion of but one or two hours. The test should be carried over several days. The hyperphoria thus uncovered should furnish a basis for rational treatment. It becomes evident that although the patient has a corrective power to overcome, perhaps even high degrees of vertical displacement in exceptional instances, such correction can only be brought about by great effort, which accounts for the asthenopia.

In the rare cases in which considerable degrees of hyperphoria do not seem to be uncovered by the usual tests, but prolonged occlusion discloses the fact, it is probable that the constant effort of the patient to maintain a common level of the images, becomes more or less automatic in spite of the dissimilarity of the images created by the Maddox rod. The corrective measure becomes a subconscious one. Prolonged occlusion, for the time being, breaks in on the habit, and the true amount of hyperphoria is therefore made manifest.

This same corrective power is occasionally found after prism training in exophoria.

Treatment.—The treatment of hyperphoria as in all forms of heterophoria is essentially individual. Aside from the careful correction of the refractive error, no fixed rules can be laid down as applicable to every case. In a general way, however, one can point out certain underlying principles, which if carefully followed, will lead to satisfactory results in a majority of instances.

As refractive errors are in themselves a source of asthenopic symptoms, independent of, or associated with muscular asthenopia, it is important that these errors should be carefully corrected, under proper cycloplegia when possible. A careful muscle balance should be taken with the correction on. If hyperphoria of a moderate degree, 1 diopter or under, is found, the patient should wear his correction and be asked to return in about two weeks for further treatment or observation. One prism diopter of hyperphoria can be overcome by the average patient without any inconvenience. Occasionally, however, even 1 diopter may cause symptoms which can be relieved by a correcting prism.

Correcting Prisms in Glasses.—The logical step in the way of relieving asthenopic symptoms due to hyperphoria, if the proper correction of the ametropia has not brought relief, is the prescribing of corrective prisms to be worn constantly with the ametropic correction. To the thoughful student, several questions naturally arise. How low an error should be corrected by prisms? How high an error can be corrected by the same method? How much of the hyperphoria should be corrected? How should prisms be placed in the frames for constant use, and we might add a final question, does the wearing of prisms in the vertical increase the hyperphoria?

The last question has been asked so frequently that it should receive consideration first. The wearing of prisms for the correction of hyperphoria *does not increase the trouble.* After hearing the *pros* and *cons* on this subject, one can hardly refrain from being more or less dogmatic. It is true that a patient in whom we may uncover 2 diopters of hyperphoria today, may return in three months after wearing a correcting prism, with 1 or even 2 diopters more. The author recalls a number of patients of this type, but one in particular, in whom additions had to be made during the course of a year until 5.5 prism diopters were incorporated in her glasses. And not until this total amount of hyperphoria was uncovered and corrected did the patient obtain relief. The question naturally arises, did the prescribing of the prisms increase the hyperphoria, or was the hyperphoria latent and if so, why was it not uncovered at first? The hyperphoria was latent in this case, as it is in many other cases. Failure to uncover the total amount at the first or at succeeding examinations was due to an unusual faculty on the part of the patient to hold her hyperphoria in check, a faculty which of necessity became a habit with her As prisms were added to her correction, the necessity for her constant effort to hold the hyperphoria in check, became less, and her latent hyperphoria gradually became manifest and was corrected, with entire and permanent relief of her asthenopia. It should be added that each addition to her prismatic correction gave temporary relief but after a time the symptoms returned.

It occasionally happens that a patient will return in a year or two after wearing prisms for the correction of hyperphoria. No appreciable amount of hyperphoria can now be uncovered and it becomes necessary to remove the prisms. This may lead to the belief that the original measurement of the hyperphoria was in error or that the wearing of the prisms corrected the existing vertical imbalance. It is probable, however, that neither of these explanations is correct. The error, if any, was probably one of judgment in the first place in prescribing a prism in the vertical before the refractive correction was worn sufficiently long to determine whether this correction alone might bring about a perfect balance in the vertical. As was pointed out earlier in the discussion of heterophoria, the correction of a refractive error may lead in certain instances to a return to normal muscle balance. It is a rule well worth following, to withhold prisms in the vertical, in low degrees of hyperphoria, until the ametropic correction has been worn for several months. If hyperphoria continues and the patient has manifest symptoms of the same, the time has arrived for the prescribing of prisms.

How low an error shall be corrected by prisms? There is some difference in opinion on this point, as there is even today on the question of the maximum amount of ametropia to be corrected. The answer depends largely upon the patient and the symptoms of which he complains. The average individual will rarely suffer an appreciable inconvenience from 1 diopter of hyperphoria. Occasionally, however, a patient with weak vertical duction power becomes conscious of the effect of even 1 diopter and is at once relieved when a correcting prism is prescribed. It is difficult to draw a sharp dividing line as the question becomes one of individuality. It is the author's practice to regard a hyperphoria of 1.5 diopters as of sufficient importance to correct even at the time of prescribing glasses.

How high an error can be corrected by prisms placed vertically? It is rarely expedient to incorporate more than 10 diopters in the two lenses, 5 before each eye. When the vertical error exceeds this amount to an appreciable extent, it is better to consider operative measures for the correction of the same.

How much of the hyperphoria should be corrected by prisms is an interesting question. It is generally thought that two-thirds of the amount should be corrected. It is a "rule of thumb" which does not always work to the patient's comfort. If the patient has not previously worn glasses, it is not unwise to correct two-thirds of the hyperphoria. If, on the other hand, the patient has worn glasses even approximately correct, it is the author's experience that a nearly full hyperphoric correction is gratefully accepted. For example, a patient showing 4 diopters of hyperphoria, will accept 3.5 for constant use in prismatic correction.

It rarely happens that the same degree of hyperphoria is found at 6 meters and at the near-point. Two and a half for distance and a corresponding 2 for the near-point or *vice versa* is not unusual. If repeated examinations have shown this relative proportion to be correct, it is best to prescribe the full minimum for constant use. Occasionally one finds a considerable difference in the degree of hyperphoria at 6 meters and at 33 cm. When the greater degree obtains at the near-point, and the patient is employed at close work throughout the day, it may even become necessary to prescribe two pair of glasses with the proper prismatic correction in each pair of glasses. If bifocals are worn and the near-point hyperphoria is much in excess required for distance, a "hook front" containing the additional prism correction for the near-point may be prescribed for prolonged close work.

How shall prisms be placed in glasses for constant use? A prism of 1 diopter or less should be placed base down before the hyperphoric eye. When higher degrees are needed, the amount should be divided between the two eyes, one-half the amount placed base down before the hyperphoric eye, and the other half *base up before the hypophoric eye.* It would seem that this method of placing prism in frames would not need emphasis. Experience, however, is to the contrary. The hyperphoric eye is higher than its fellow and its image is lower. The prism should be so placed that the low image is raised, hence base down. The hypophoric eye is lower than its fellow and its image is higher. The prism is placed base upward in order to lower its image.

The division of the prisms equally between the two eyes is not always necessary. One can be guided by the relative sphero-cylindrical correction and the greater amount of prismatic correction may be placed in the weaker lens in order to balance the weight of the two lenses. As we are dealing in low-diopter strengths of prisms, the color aberation and distortion effects of prisms of high degrees need not be considered.

Prism Training in Hyperphoria.—This method of treating a tendency to vertical deviation has proven to be unsatisfactory. The same may be said of the role of the stereoscope in hyperphoria. It is a significant fact, that the vertical imbalance is present early in life, although permanent symptoms of the same may not appear until the age of maturity. Furthermore, throughout life there is little variation in the vertical duction power of the eyes. Supraduction and infraduction are rather definite in the same individual. On an average, there is no great variation in different individuals. But rarely does one find a supraduction or an infraduction power of more than 3 diopters. On these grounds alone, theoretically one would not hope for much from the standpoint of training. In practical experience little is accomplished. The use of the stereoscope probably accomplishes some good in temporarily relieving symptoms, but in the author's experience even the stereoscope fails to furnish permanent relief.

Medical Treatment.—Medical treatment is of little avail in a well-established case of hyperphoria. In persons of a neurotic temperament, or in persons leading a tense life, the asthenopia may be periodic in character. In such individuals relief may be obtained during the periods of exacerbation by paying attention to general health measures. Sedatives may bring temporary relief in the overactive, or tonics may be indicated in the undernourished. One should not overlook the influence of the use of tea, coffee, and tobacco. While tea and coffee and the use of tobacco do not have any direct bearing upon the production of any of the phorias in many individuals, they are capable of aggravating or even of precipitating symptoms of asthenopia. The relief from symptoms when tea and coffee, or even tobacco, have been withdrawn, has led the author to make careful inquiries as to habits in all marked cases of heterophoria. Occupational influences may also be checked by rest. All these measures, however, are only palliative and should be followed or replaced by rational methods which will bring permanent relief.

Operative Treatment.—In a majority of instances, proper correction of refractive errors and the prescribing of prisms in correcting glasses for any residual hyperphoria, are so satisfactory that operative interference need only be considered in high degrees. When surgery becomes necessary, the type of operation and the muscle or muscles which are to be operated on should receive careful consideration.

As a rule complete tenotomies should not be practised. The danger of marked over-correction or of the creation of a paralytic squint is always present. A case in point will illustrate this danger. For some years past, the author has had under his care a patient who had 10 diopters of left hyperphoria, for which the surgeon in charge performed a tenotomy of the left superior rectus. The result was the conversion of 10 diopters of left hyperphoria into a 10 diopter right hyperphoria with weak rotational power of the left eye upward. In the same patient, an exophoria was converted into a paralytic squint of the left external rectus by a tenotomy of this muscle. The effect of these tenotomies upon the general health and the mental upset of this patient for seventeen years subsequently would be sufficient to convince any surgeon that this type of operation should be replaced by a more rational method. It fell to the author's lot to reattach the external rectus with fair restoration of lateral balance and recovery of the patient's health. The right hyperphoria was relieved in part by correcting prisms.

Modified or partial tenotomies accomplish little in lengthening a tendon and in addition to inefficiency, there is danger of producing torsional effects which are equally serious.

In the author's judgment operative correction of hyperphoria should be limited to modified advancement or tucking of the opposing muscle or the setting back of the over-acting tendon. In the latter operation, great care should be observed in the exact placing of the tendon with a due regard for the line of the previous attachment in order to prevent subsequent torsional action. Operations on the vertical muscles are more difficult than on the lateral recti, and general anesthesia will aid the operator in carrying out minute details better than when performed under local anesthesia. When set back, the tendon should be reattached to the sclera and firmly anchored. Conjunctiva and capsule should be disturbed as little as possible. If the operation is properly performed, the results are satisfactory.

Either a modified advancement of the inferior rectus of the hyperphoric eye, or of the superior rectus of the hypophoric eye, if the upward rotation of this eye is shown to be weak, is equally satisfactory. Operations on the inferior rectus have an additional advantage in the fact that the muscle is more accessible. In the type of advancement referred to a small incision is made in the conjunctiva 8 mm. from and parallel to the cornea. The tendon is exposed and the central three-fifths of the tendon is freed from its attachment. This central portion is grasped by fine-tooth forceps, and incisions are made on either side for a distance of

4 mm. A No. 1 twisted double-armed suture is introduced by a whip stitch into its outer and inner thirds, from without in. The needles are now introduced into the episcleral tissue, brought out through the conjunctiva near the limbus and the sutures are tied. The conjunctival wound is brought together by a continuous suture. Both eyes are closed for four or five days as in the Worth operation. The operation has points of similarity to the operation of Stevens which he called "tendon contraction operation." It differs materially from this operation in that the tendon is advanced and firmly anchored to the sclera, instead of to the conjunctival tissue as practised by Stevens.

The points of advantage are: (1) It counteracts the over-acting superior rectus by giving the inferior rectus better purchase. (2) The inferior rectus is more accessible and furnishes a better operative field than the superior muscle. (3) The sclera is thicker toward the limbus than it is just posterior to the muscle attachment, and better anchorage is thus obtained. (4) If through faulty technic, the advanced part of the tendon should not hold, the intact outer and inner fifth, will not admit of much if any recession, and failure cannot bring about a worse condition than existed previous to the operation. Any residual hyperphoria, left after the advancement operation, usually falls within the range of correcting prisms.

The cinch operation, as now practised by Roderic O'Connor of San Francisco, Cal., as a means of shortening a weak opposing muscle, should receive consideration, because of the accuracy of the amount of shortening which can be obtained by this method. The degree of shortening can be estimated with fair accuracy by the number of strands of the dermal sutures which are employed.

Tucking of the inferior or superior rectus is more difficult than that of the external rectus. Those who are partial to tucking, however, will find this method of shortening a satisfactory procedure in hyperphoria, if a shortening of a weak muscle is indicated.

Before operating, the upward and downward rotations of both eyes should be taken. If any defect in rotations is found, the operation should be directed to the strengthening of the muscle of weak rotation. If no defect in rotations is found, the operation should be directed to the downward pull of the hyperphoric eye.

ESOPHORIA.

Esophoria is a tendency for the eyes to deviate inward. As in all forms of heterophoria, the deviation is held in check in the interest of single binocular vision. The constant effort required to overcome the converging tendency, and in part the confusion which must necessarily follow a failure of the two images to promptly fuse, are responsible for the symptoms which arise in this condition.

Etiology.—An analysis of large numbers of cases of esophoria leads to interesting deductions as to the underlying factors. In studying large groups, one is struck with the relative infrequency of esophoria of high degrees, as compared with exophoria. An esophoria of 1 or 2 diopters is found in a large number of cases when the muscle balance is taken at 6 meters. In fact, in routine muscle studies, a small degree of esophoria is the rule, and one might say normal. It is only in those cases in which an increased amount of convergence is noted for the near-point, or a high degree of esophoria is present at infinity, that the condition becomes serious and gives rise to symptoms.

The refractive error present is a conspicuous factor. Hypermetropia with hyperopic astigmatism is observed in a large number of cases. This leads one to regard the accommodative group of esophoria as dominant. Attention has been called to the intimate relation between the diopter of accommodation and the meter angle of convergence. The importance attached to the rule of prescribing as full a hyperopic correction as the patient will tolerate, and the stress laid upon accommodation and convergence repression also emphasize the fact that the refractive error is an important etiological factor. Not all patients, however, who are victims of esophoria are hyperopic. Some have low degrees of myopic astigmatism, and others have considerable degrees of myopia. Other causes, therefore, are responsible.

In contrast to exophoria, esophoria occurs in the strong or sthenic type. It is a condition of over-action, especially over-action in a highly neurotic type of individual. In fact, if one were asked to name the second great factor in esophoria, the answer would unhesitatingly be a "highly strung" nervous system. In esophoria of high degree, with marked reflex symptoms, whatever other factors may contribute to the condition are over-masked by an outstanding unstable nervous system. These are the difficult cases to manage, not only because the nervous system is lacking in balance, but because this very condition does not bespeak a whole-hearted coöperation of the patient, which is most important in treating this discouraging type of heterophoria. Again an inherited instability is usually whipped into greater excitability by the use of tea, coffee and tobacco. The author has repeatedly been able to convert an aggravated form of esophoria into one of moderate degree by simply cutting off a vicious habit of using tea, coffee or tobacco in this type of patient. In the same catagory as excitants should be mentioned the improper functioning of the ductless glands. An esophoria of 14 to 18 diopters, without any stability, which occurred in a young woman, aged twenty-five years, after a double oöphorectomy, was restored to normal balance and so maintained by glandular therapy and the use of the stereoscope. The factors just mentioned would probably not be operative in individuals of good nervous balance, but act as exciting factors in those who may be said to be of neurotic temperament.

In the same type of case, occupational influences are especially noticeable. The high tension methods of modern business, the handling of large numbers of men, or the responsibility involved in directing industries on a large scale, to say nothing of the minor vexations and difficulties encountered by those in less responsible positions, all tend to create a nervous imbalance in most individuals unless they happen to be of a phlegmatic temperament. This nervous upset is apt to become manifest in the production of esophoria which is difficult to manage because the average business man, embarked on an industrial program of the type to which reference has been made, cannot see his way clear to change his habits without relinquishing his hold on his business.

Over-action on the part of some of the ocular muscles should be considered. This has been assigned as a cause of esophoria. It is probable that overly strong internal or abnormally weak external rotators, may be the underlying factors in a limited number of cases. It should be remembered, however, that with few exceptions, excessive convergence or deficient divergence are innervational. Many of the so-called sthenic cases of apparent over-action of convergence are simply the result of undue innervation with inability to hold convergence within limited bounds. When this condition remains over-active for long periods, the convergence tendency becomes fixed or even intensified. Whatever role over-acting interni may play in esophoria, such over-development should be regarded as the result of innervationsand not primarily an intrinsic over-strength of these muscles.

In the consideration of esophoria and exophoria, the student should bear in mind that although the *interni* are the leaders in convergence, and the *externi* play the same dominant role in divergence, it is the combined action of synergists and antagonists, under the influence of innervations and the governing action of the fusion faculty, that brings about the results. It is not the action of individual muscles. This statement is entirely consistent with the surgical treatment of the condition, even though surgical treatment is applied to single muscles. This will be restated in the surgical treatment of these conditions.

The cause of esophoria, in a given case, is, as a rule, to be found in one or more of three groups of factors. They are: (1) Muscular; (2) accommodative; (3) innervational. The first group are rarely operative, excepting in a secondary way. Muscular anomalies, such as over- or under-development, or faulty attachments, are the exceptions, and therefore rarely operative. Apparent over-action or under-action as a rule are the result of excessive innervation and a faulty fusion faculty. One cannot, however, lose sight of the fact that there may be an inherent defective relation between the internal and external rotators, and because of this disturbed balance, the convergence innervation may be in excess, resulting in esophoria, or it may be deficient and exophoria may develop. A careful study of prism abduction and adduction will determine the relative ratio. As previously pointed out, they should be in proportion of 1 of abduction to 3 of adduction. Both may be defective, or both may be excessive, but as a rule if the relative strength of 1 to 3 is approximately maintained, lateral disturbance is not of great consequence, and any existing esophoria cannot be attributed to muscle defect. On the other hand, marked disturbance of the 1 to 3 ratio, found in rather high degrees of esophoria, may or may not be due in part to excessive muscular action.

The accommodative element is easily determined. It is active to some extent in practically all cases of esophoria, whether high or low, when a considerable amount of hyperopia is present and uncorrected.

The group included in the term "innervational" usually includes a majority of cases in which the accommodative factor is not sufficient to account for the esophoria. It is difficult at times to determine how much of convergence excess is due to this element and how much should be attributed to a lack of balance in the duction strength existing between the external and internal rotators. However, in the management of these cases, it is safe to assume that the difficulty is largely innervational and not muscular. At all events, in the author's experience, the treatment which has been found to be most effective in this type of case, namely stereoscopic training, tends to correct and stabilize faulty innervation and pure muscular imbalance as well.

Symptoms.—The symptoms of esophoria are local and general, or ocular and reflex. The conjunctiva becomes red and the lid margins show congestion even to the extent of marked *blepharitis marginalis* with thickening of the lids, and the formation of scales on the lid margins, pustules, and the falling out of lashes. In the milder cases, any effort at near work may cause a burning or smarting of the conjunctiva, with epiphora. Repeated or prolonged congestion may result in occlusion of the puncta. These local conditions are not peculiar to esophoria, but are evidence of eye strain such as may result from other forms of muscle imbalance and to the strain incident to refractive errors.

Associated with this local evidence of asthenopia, and even of more serious consequence, are the general or reflex symptoms. Attempts at reading cause the print to run together, and the patient becomes drowsy. Headaches, supra-orbital and temporal, of the panoramic type are frequently experienced in crowds, in railway carriages, and in automobile riding, in attending moving pictures, or on shopping expeditions, as well as after using the eyes in close work. Instead of headache, or associated with headache, there may be marked gastric disturbance even to the extent of vomiting and the loss of weight. The cause of these reflex symptoms is found largely in the effort required to hold in check the blurring of images, by the tension of the opposing or antagonistic group of muscles. So long as the condition remains as an esophoria, diplopia is held in abeyance, and the images are fused by great effort. It is the effort, therefore, which wears on the individual and brings about varied reflex phenomena. When the patient is no longer able to fuse, the tendency becomes a manifest squint with relief from the symptoms just described.

In studying aggravated forms of esophoria, with marked disturbance of the nervous system, it becomes difficult at times to determine whether the esophoria is the result of a badly upset nervous system, or the nervous disturbance has been caused by the esophoria. Either is possible. It is the author's experience, however, that the esophoria is usually caused by the general nervous irritability of the patient, and that an improvement is noticeable in the ocular muscle imbalance when sedative measures are employed to stabilize the general nervous system. At the same time, an aggravated esophoria may act as a reflex cause in unbalancing the nervous system.

Treatment. The presence and degree of esophoria is determined, by one or more of the methods described, at infinity and at the near-point. Abduction and adduction are carefully measured, and the convergence near-point is accurately estimated as a routine preliminary study in every case of esophoria. With this data clearly in mind, the surgeon proceeds in a logical manner to overcome the trouble in the following order.

Refraction —Inasmuch as the relation of the diopter of accommodation to the meter angle of convergence is of the greatest importance, the first logical step in the treatment of esophoria is to measure the total refractive error under a suitable cycloplegic. The selection of the cycloplegic is of much importance. If the patient is known to be a myope, homatropine, when properly employed, may be sufficient to uncover all latent errors. In the hypermetrope, with an especially active ciliary muscle, it is the author's experience that homatropine is unsatisfactory. In children, a 1 per cent atropine sulphate solution should be employed, and the examinations should be repeated until the surgeon is satisfied that the total astigmatic and spheric error has been properly measured. In adults up to the age of forty-five years, a solution of scopolamine hydrobromate $\frac{1}{4}$ per cent, to which is added $\frac{1}{2}$ per cent of cocaine hydrochlorate, is most satisfactory. Three instillations at intervals of fifteen minutes are usually sufficient to insure complete cycloplegic action in one hour after the first instillation. This same process should be repeated on the second day and a second examination by retinoscope and trial case should confirm or correct the error found at the first examination. An objection raised by some against scopolamine is the danger of toxic symptoms. In the author's experience, this objection is without foundation if the instillations are made in the office and by one who is qualified. If the precaution is taken to compress the puncta for a few moments after each instillation, no poisonous symptoms will appear. This is a precaution which should be followed in the use of atropine as well. If the patient complains of dryness of the throat or of slight dizziness, a small dose of aromatic spirits of ammonia affords almost instant relief.

Having obtained the total latent refractive error, after the effects of the cycloplegic have worn off, the surgeon is in a position to prescribe glasses. In hypermetropia, the fullest correction which the patient will tolerate by the fogging method, should be prescribed for constant use. In adults, it is always unwise to prescribe a full correction while the eyes are under the influence of the cycloplegic, and it is equally unwise and unscientific to cut a cycloplegic correction by a "rule of thumb." It may be possible to force children into the habit of wearing a full correction, but the same rule cannot be applied in every instance in adult life. If the post-cycloplegic test is made by the fogging method, after the *full error is uncovered under a cycloplegic,* so as to insure complete correction of the astigmatism and perfect balance of the spheric error, 0.25 to 0.50 diopter more of the remaining spheric error can usually be added for binocular use. In fact, if the spheres are added 0.25 diopter at a time before both eyes, the patient can be gradually trained to tolerate a moderately full correction. This method also insures that it will be worn. On the other hand, if the full correction is prescribed under the cycloplegic, without a knowledge of the patient's tolerance, the chances are, the patient will not wear his correction, and the surgeon will be defeated in the most important step in the correction of the asthenopia. It should be remembered that for every spheric diopter worn, there should be a correction of 1 meter angle of convergence. Therefore, the fullest correction which the patient will tolerate and *wear* should be prescribed. In myopia, a full correction should be prescribed, although it may have no influence on the correction of the esophoria.

If the esophoria is purely of the accommodative variety, a full correction of the hypermetropia usually brings relief to the patient. In this type of esophoria, there is a higher degree of excessive convergence for the near-point than there is at 20 feet or 6 meters. If a full correction for distance will not be accepted by the patient, a second pair of glasses should be prescribed for close work in which the full spheric error will usually be tolerated. This will give relief when it is most needed. If the patient is taken into the surgeon's confidence, and the needs in his case are made clear to him in simple language which he can understand, few people fail to give hearty coöperation in the handling of their case in an unusual way. It is surprising too when two pair of glasses are thus prescribed, how rapidly the patient finds he can wear his strong glasses with increasing comfort and how soon he will discard the weak pair for the stronger ones for constant use. If the surgeon is convinced that any existing esophoria is due solely to the accommodative factor, every intelligent effort should be made to correct the excessive convergence by means of glasses. The average patient will gradually accept more and more of his hyperopic spheric glass with relief from his asthenopia. If the ciliary muscle remains too active and objects at a distance tend to blur, the method of using very weak solutions of homatropine, scopolamine, or atropine, are to be recommended. Until the patient has clearly demonstrated his inability to wear his full correction by the methods advised, the other steps in the treatment to be described should not be practised.

Medical Treatment.—After glasses have been prescribed, the next step in the management of the case is directed to the patient's general condition. The elimination from the diet of tea and coffee often becomes necessary in patients of nervous tendencies. The same applies to the excessive use of cigarettes or other forms of tobacco. A healthful, outdoor life, with proper exercise, helps to stabilize an irritable nervous system in bodies of strong physique as well as in the weak. Rest from confining and tedious occupations, and at times complete cessation of work at the near-point, are of great help in restoring nervous equilibrium. There are times when a prolonged restful but healthful vacation spent largely in the open air or on board a ship will accomplish a great deal in restoring an irritable nervous system to a normal balance. All these measures may be necessary in aggravated cases. Nerve sedatives may be employed when the conditions present seem to indicate their use. In an average case, they probably do not accomplish much. In selected cases, however, they may be indicated, and should be used.

Prism and Stereoscopic Exercises.—In the use of prisms, one aims to increase the abducting power of the external rotators, or more correctly speaking, to strengthen divergence. It has been clearly shown that little can be accomplished in this direction by the use of prisms at 6 meters. If the abducting power is found to be low and out of proportion to the adducting power of the patient, it is a good method of procedure to at least attempt abduction training. The method is as follows: Prisms of low power are held, bases in, before the patient's eyes as he fixes on a small muscle light 6 meters or 20 feet across the room. If abduction has been found to be 4 prism diopters, for example, a 2 diopter prism is placed base in before one eye. If the two images can be fused, a 2 diopter prism is added, base in, before the other eye. If the two images fuse, an increase in the prism strength of 1 diopter at a time is made until the maximum point of fusion has been reached. The results, as a rule, are discouraging. One can determine in a few attempts, in the office, whether the patient will respond to treatment. If the response is good, daily practice at the patient's home under the same conditions should be added to the office training.

In the author's experience, prism training in esophoria, as in hyperphoria, is disappointing. On the other hand, stereoscopic exercises have been found to be of considerable value. In a majority of cases, excessive convergence is due to the inability of the patient to hold the innervations within bounds. The response to the innervation to converge is excessive with inability on the part of the patient to hold it in check. The stereoscope with the aid of fusion tends to stabilize and to restrict excessive action. Furthermore, convergence and divergence are moderately exercised if the chart is slowly moved back and forth along the arm of the stereoscope. In addition to the ordinary use of the instrument, according to Wells, its value can be much enhanced by placing additional prisms in front of those used in the stereoscope. In fact, Wells feels that a "phoro-optometer stereoscope" is necessary to fully carry out his method. In the Wells' model, which is a modification of that of Dr. Richard Derby, it is possible by an endless screw to separate and approximate the images. Instead of the "phoro-optometer stereoscope" of Wells, the author attaches spring clips back of the lenses to hold loose prisms, placed base in. The patient is instructed to begin with 1 or 2 diopter prisms, and the strength is slowly increased as the exercises progress. The cards are moved slowly back and forth along the stereoscopic arm, as in the exercises described. This method of increasing the abducting power by prism and stereoscope combined yields better results than attempts at prism training at 6 meters.

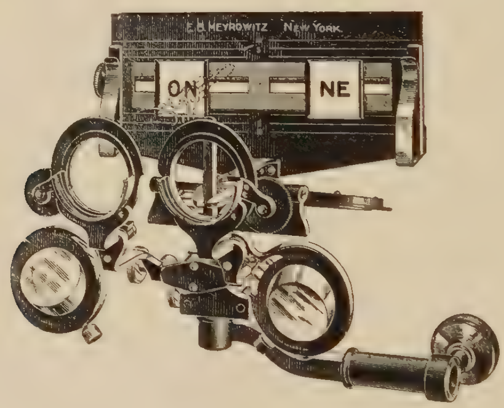


FIG. 60.—The Phoro-optometer stereoscope with adjustable object carrier. (From Wells' "The Stereoscope in Ophthalmology.")

Those who are especially interested in Wells' method, are referred to his book on *The Stereoscope in Ophthalmology.*

There is this objection to the simple use of ordinary stereoscopic photographs. Unless the patient is an intelligent adult, who is thoroughly coöperative, one cannot be sure that stereoscopic vision is maintained at all times. It is always best, therefore, to use the fourth edition of Wells' charts for the purpose. One can explain in detail what should be observed and the patient is less likely to fail to fully coöperate. In the experience of the author, patients vary much in their ability at first to obtain full stereoscopic vision. The patient is instructed, therefore, in the office to practise first with charts which offer the least difficulty, and as the practice progresses each day, new charts can be added.

In the accommodative variety of esophoria, which does not yield fully to a proper correction, the stereoscope has helped the author to make the close work of many esophorics comfortable. It is, however, in those cases in which the neurotic element is pronounced, and the esophoria varies much from day to day, that the author has found the stereoscope of greatest value. A woman, aged twenty-five years, profoundly neurotic with an irregular esophoria amounting almost to an esotropia at times, was made entirely comfortable in her work as secretary, and muscle balance was completely restored by the persistent use of the stereoscope. It required several months of work, but the results were most brilliant and justified the efforts of both patient and physician.

Convergence and Accommodation Repression The best method of practising convergence repression is by the prescribing of a full hyperopic correction when it is accepted by the patient. It is a well-known fact, however, that many patients, suffering from high hyperopic errors, who have not worn glasses for many years, will not accept a full correction. As previously pointed out, the prescribing of a full correction for near work occasionally solves the problem in that accommodation is reduced to normal by such glasses, and convergence excess is at the same time repressed.

Hansell and Reber, and others, have recommended the use of the addition of 1 or 2 diopter spheres to be worn each day for close work to suppress accommodation and thereby influence excessive convergence. While the author's experience with this method has not been very happy, others claim to have obtained good results in a limited number of cases. The chief objection to the method lies in the fact that most of these patients are more or less neurotic in temperament. The method in itself is irritating to patients because they find it difficult to relax their accommodation. Anything which will increase irritability will also increase instability in their muscle balance, and in a majority of patients, the author found it necessary to abandon the practice.

Prisms Incorporated in Glasses. The distressing symptoms in certain types of esophoria, which are not relieved by the methods of treatment thus far discussed, will demand further consideration.

It is generally agreed by those of broad experience that prisms worn with the correction are of little aid in relieving esophoria. One is confronted at times with the necessity of at least trying the method. A 1.50 to a 3 diopter prism may be ground into the glass before each eye, the prisms being placed base out. As the test is largely experimental and not likely to succeed, the author usually prescribes the prisms in the form of a "hook front" to be attached to spectacles. If such addition to the glasses is found to be comfortable and of value, they are later ground into the correction either for constant use or for near work only, as the case may be. When needed only for near work, the "hook front" offers the most satisfactory form for use. The practice has little to commend it. It may be reserved as a last resort for patients who will not coöperate and at the same time will not consent to surgical relief.

Operation for the Relief of Esophoria.—There are instances in which surgery offers relief to high degrees of esophoria when other methods of treatment are of no avail. Before resorting, however, to any surgical procedure, the conscientious operator will study most carefully the type of case with which he is dealing, and the method of operation which offers the greatest hope of success. Patients of a highly neurotic temperament with varying degrees of esophoria from day to day, which cannot be stabilized by the conscientious use of the stereoscope, are not suitable subjects for surgical interference. Esophoria, however, of high degree and rather constant in value at each examination, can be relieved by well-chosen surgical measures when other methods of treatment fail.

Partial Tenotomies.—Partial tenotomies are of no value. Some forms of partial tenotomies tend to develop a cyclophoria and thus only add to the patient's discomfort. A second cause of failure lies in the fact that unless the entire tendon is detached, a remaining strand, although slight, may be sufficient to maintain the power of the muscle. This fact surgeons have learned by experience in their failure to correct squint in the days when tenotomies were regarded as sound methods of procedure. The leaving of a single strand is sufficient to render a tenotomy useless. To this fact Lancaster adds his belief that each ocular muscle possesses potential kinetic energy far in excess of its needs.

A third, and the chief cause of failure of a partial tenotomy to relieve esophoria, is the important fact that the condition is innervational in origin. In order to overcome excessive convergence the internal recti should be so placed that, as leaders in the synergistic group, which brings about convergence, the mechanical strength of these muscles is reduced, or the opposing group should be strengthened. This cannot be accomplished by a partial tenotomy.

Full Tenotomies.—The dangers of full tenotomies are well known. The creation of a paralytic squint or of a concomitant squint, divergent in character, is only too frequently the result of complete tenotomy of an internal rectus. Furthermore, even though the tendon may become reattached, few patients upon whom tenotomy of an internal rectus has been performed, are able to sustain convergence at the near-point for comfortable work thereafter. The author recalls the case of the wife of a physician whose internal recti were tenotomized for high esophoria. The result was an exophoria of higher degree and inability to converge to a near-point working distance. Judging from the number of cases of this type seen in daily practice and the cases of paralytic squint caused in the same manner, one would conclude that it is unnecessary to raise a note of protest against so unsurgical a procedure Clinical experience, however, confirms the necessity for such protest.

Recessions, Resections and Tucking Operations.—These are the constructive surgical methods of correcting high degrees of esophoria. As to the relative value of each, there may be some difference of opinion. The author's preference is stated in the order as given and the reason for the same will be given.

Measures which will bring permanent relief to this condition are either the setting back of the interni or shortening of the externi. As to which is indicated is largely determined by the prism duction power of the internal and external group. Clinically one finds most frequently an excessive convergence, less frequently a divergence below the normal. If divergence is fair and convergence is greatly in excess, some form of recession operation practised upon one or both internal recti is logically indicated. If divergence is much reduced and convergence is not greatly increased above an average normal of 18 to 20 prism diopters, a resection or a tucking of one or both external recti is indicated. An advancement of an external rectus is rarely indicated in esophoria.

In addition to this method of determining whether the interni shall be weakened, or the externi strengthened, the discriminating surgeon will consider certain details in the two types of operations and their probable bearing upon ultimate success. Recession of an internal rectus to any fixed point on the globe from 1 to 5 mm. back of the tendon insertion, can be performed without risk of stitches cutting through because of an absence of tension. Recovery is rapid and occlusion of both eyes need not be practised so long as when a resection is performed. A resection is accompanied by tension on the sutures, and requires occlusion of both eyes for a period of at least five days. Recovery is more prolonged than in a recession operation. If the sutures should cut through and the tendon should slip as a consequence, the esophoria may be increased. For the reasons given above, in addition to the fact that in esophoria we are dealing as a rule with a sthenic condition, the author's choice is in favor of recession of the interni rather than resection of the externi.

Resections, or Tuckings.—When preference is given to an increase in the abducting power rather than to a decrease in adduction, the choice of operation will be a resection or a tucking of one or both externi. Resection with advancement is rarely practised because as a rule either of the operations in question are sufficient to overcome considerable degrees of esophoria. The choice is largely one of personal preference. Many believe that a tuck in the external rectus is safer, more easily performed, and equal in value to a resection. If properly performed this particular indication for a tucking operation is the ideal one. As a rule, in the author's judgment, tucking should be limited to cases in which a moderate increase in power is desired. It is, therefore, ideally adapted to the correction of this form of error. The objections to tucking will be dealt with more fully in the section on Concomitant Squint. The fact that the muscle need not be cut appeals to patients and often to the surgeon. A resection, however, is of equal value and is preferred by many. The cinch method of shortening, as practised by O'Connor is ideally adapted to the relief of esophoria, if increased divergence is indicated. Whatever the method of choice may be, the young operator should always aim to over-correct. The average result is less than one expects or hopes to obtain.

Shall the surgical procedure be divided between the two eyes or limited to one? Unless the esophoria is very high, surgery may be limited to one eye. Ideally a double recession or a double advancement is indicated, but this also exposes the patient to the double danger, though slight, of infection, and the other increased risks of a second operation. Preferably, one muscle should be operated upon, and if the results are insufficient, the corresponding muscle in the other eye may be operated upon at a later date.

EXOPHORIA.

A tendency for the visual axes to deviate outward is probably encountered in practice more frequently than hyperphoria or esophoria. In youth and early adult life up to thirty years of age, it is of comparatively rare occurrence, but after thirty years, the number of cases increases rapidly until presbyopia becomes fully established, when it is observed in excess of either hyperphoria or esophoria. The student may recall that hyperphoria is as common in adolescence as it is in early- and middle-adult life. In a majority of instances it continues unchanged throughout life, although its presence may not give rise to marked symptoms during early adolescence. When established, it is a rather fixed quantity. To a less extent, esophoria is a condition which is observed frequently before twenty years, and after thirty years diminishes in frequency rather than increases.

Hyperphoria is a rather constant and an inherent weakness in the muscle balance which may be observed in youth if sought for, although its symptoms may not attract attention until close school work is begun. Esophoria is present, as a rule, during the adolescent period, when muscular energy and nerve force are at their best, and more or less unrestrained either because of excess or because of instability of the nervous system. Exophoria develops at a later period in life, and while not an indication of weakness or of declining power, it is at least a passive condition rather than active. It is intimately associated with a deficient or declining accommodative power in a large number of cases—accommodative exophoria, as observed in myopia and in presbyopia, but it is also present in hyperopia before the presbyopic period. Hansell and Reber in a series of 441 cases of exophoria found hyperopia with astigmatism present in 72 per cent. These figures are rather higher than the author's experience. They serve well, nevertheless, to illustrate the point that certain factors, other than accommodation, are responsible for a number of cases.

Etiology.—It is difficult to group clinical types of exophoria so as to include all etiological factors. The following classification is fairly comprehensive:

1. *Accommodative exophoria*, a type of exophoria which is associated with and largely dependent upon myopia with astigmatism.
2. *Essential exophoria*, a type which may be associated with hyperopia and astigmatism and occasionally with myopia.

Subdivisions of this group, following Hansell and Reber, are:

(*a*) Exophoria, in the presence of hyperopia and hyperopic astigmatism with normal divergence and deficient convergence.

(*b*) Exophoria, with hyperopia and hyperopic astigmatism in which divergence is excessive and convergence is normal.

(*c*) Exophoria, with hyperopia and hyperopic astigmatism in which convergence and divergence are deficient.

(*d*) Exophoria, with myopia, in which a convergence insufficiency has increased to such an extent that correction of the refractive error cannot overcome the divergence tendency.

3. *Exophoria of Presbyopia.*—It will be noted that the first and third groups have what might seem to be a common cause. Failing accommodation, however, in presbyopia differs materially from the defective accommodation found in the first. The treatment of these groups requires different methods and they should, therefore, be separated in classification.

Accommodative Exophoria.—The relation of the meter angle of convergence to the diopter of accommodation is definite. Uncorrected myopia admitting of the use of the eyes at the near-point without accommodation, creates a passive exophoria which may properly be called accommodative. When a full myopic correction is worn, the need for accommodation, in order to focus upon a near object, once more returns and a normal relation between convergence and accommodation is restored. This is a common form of exophoria and is easily understood. In its solution the student will find no difficulties.

Essential Exophoria.—This group is complex. It not only includes many cases of hypermetropia and astigmatism with disturbed relations between abduction and adduction, but many cases of myopia which cannot be corrected by the simple prescribing of glasses. It is true that cases which begin as accommodative types, by long neglect may develop into aggravated forms of convergence insufficiency. It is probable, however, that in addition to a passive phenomenon which plays a role in its production, the exophoria may also be due to active factors, and the author prefers to include this myopic type in the group of essential exophoria. There is in them something other than a failure of convergence because accommodation is not called into action.

In the experience of all, exophoria is found in a large group of cases in whom the refractive error is hyperopic. In some instances, divergence is in excess, in others convergence is deficient, and in a third group both are deficient. The cause is inherent and manifestly not accommodative. For the want of a better term, they may be called essential. It is difficult to assign a cause. It is probable that more than one factor is responsible. Improper muscle attachments, weak internal recti, over-developed externi, orbital malformations, variations in the development of the capsule of Tenon and the check ligaments, may all in turn have a bearing upon the development of this form of exophoria. In the treatment of this type, the surgeon encounters his greatest difficulties. Surgical interference in some form, when other measures fail, as a rule is indicated.

Presbyopic Exophoria.—Many cases of exophoria have their inception with the development of presbyopia. Two types are observed. A majority have a normal muscle balance at infinity, but, in the late thirties or early forties, develop an exophoria for the near-point as sclerosing of the nucleus develops and the power of accommodation diminishes. A smaller group develop exophoria for the near-point when reading glasses are prescribed. Both types bear a resemblance to the first group of accommodative exophoria, but have a different etiology, and therefore will be treated separately.

Symptoms.—The symptoms of exophoria are not unlike those found in hyperphoria and in esophoria. They differ with the individual and the underlying factors. Headache, as a rule, is pronounced and of the panoramic type. It is apt to be increased in crowds, in automobiling, in train riding, and by moving pictures. In many individuals it may take the form of a "nervous sick headache," and may be accompanied by nausea and even vomiting. In the author's experience, close work is more often associated with symptoms than is distant vision. Headache, nausea, eye fatigue, blurring of the print, redness of the lids, and drowsiness are the chief complaints. Hyperemia of the lids may develop into a chronic blepharitis. Drowsiness on attempts at reading, combined with conjunctival hyperemia, may become so annoying as to cause the patient to abandon all close work. Those who follow the vocation of mechanical drawing find their work particularly trying. Hansell and Reber found many of their cases suffered from neurasthenia, with all symptoms thereby increased. In the author's experience, this condition is found more frequently associated with esophoria. Age, however, is a determining factor. If neurasthenia occurs in early adult life, it is more often associated with esophoria. If it occurs after thirty years, exophoria may be associated as a coincidence rather than in a cause and effect relationship. There are instances, however, in which the characteristic general muscular fatigue of neurasthenia may be shared by the ocular muscles.

Diagnosis.—Any of the tests described may be employed in the detection of exophoria. A routine method of study should be followed in all patients whether they present themselves for refraction or for other conditions which do not include acute inflammations of the eyes and conditions in which a muscle balance cannot be taken. Routine study should include the use of the Maddox rod for far and near, the Maddox wing test or other satisfactory test for the near-point. This study at 33 cm. should be emphasized. It is not sufficient to determine the muscle balance at 6 meters. There may be orthophoria or even esophoria at 6 meters, and high degrees of exophoria for the near-point. In addition, the convergence near-point should always be taken and if exophoria is found to be present, lateral ductions are equally important. Fortified with a knowledge of the complete refractive error, the muscle balance, the convergence near-point, and the lateral duction power of the patient, the surgeon is in the possession of facts by means of which he is in a position to bring relief to a majority of these patients.

Treatment.—The results in the treatment of hyperphoria are most satisfactory as to the relief of symptoms. Cures of the condition are rarely effected excepting in those cases in which a correction of the refractive error restores the vertical balance. The treatment of esophoria is less satisfactory. Relief, however, is possible in many cases. Exophoria, on the other hand, offers a field for the interested ophthalmic surgeon, to effect cures and to bring relief to a large *clientele*. In the author's experience, there are few patients suffering from exophoria who cannot be made comfortable.

The reader will naturally ask how much exophoria should be regarded as pathological and in need of correction? What degree may be ignored as a negligible quantity? Authors speak of "physiological exophoria" meaning thereby the amount of exophoria which patients will tolerate without symptoms. The term is incorrectly used.[1] Exophoria is not physiological at normal reading distances. There is an inherent difference in patients in their ability to overcome varying degrees of heterophoria without symptoms. Again this ability may vary in the same individual at different ages and under different health and environmental conditions. The question becomes one largely of individuality. It is difficult to lay down any hard and fast rule as to the amount of exophoria which each patient will tolerate without symptoms. On an average 1 or 2 diopters of exophoria at infinite is borne by a majority of patients without complaint. Two or 3 diopters may cause no symptoms at 33 cm. *There are few patients who will not experience discomfort with more than 2 diopters for distance and more than 3 diopters at the near-point.* The degree of exophoria, therefore, which will require treatment becomes an individual one and the surgeon must be guided largely by the comfort of the patient. There is one exception to this broad statement in which the surgeon's better judgment should prevail. An exophoria, not of the accommodative variety, of 3 diopters or more, occurring in adolescence or in early adult life, should be treated even though the patient may deny any discomfort. At this early age, the patient's reserve force, supplied by his youth, may be called upon indefinitely, and no noticeable symptoms may become manifest. When, however, much exophoria is present so early in life, increasing amounts are apt to become manifest as the resistance of advancing age declines, and every effort should be made to overcome this tendency when it is first observed.

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[1] True "physiological exophoria" may be said to exist when the eyes converge to a very near-point. This is not the exophoria to which the average physician refers in using the term "physiological exophoria."

Methods of treatment will vary some in each group. One can in a way lay down general principles which will apply to all cases, variations to be made as indicated by underlying factors. With the full coöperation of the patient, the treatment of exophoria is very satisfactory. In the author's earlier experience, the accommodative type yielded promptly to approved methods, but other forms were baffling and a source of much worry. Added experience and patience combined with the awakening of an enthusiasm on the part of the patient in a matter of full coöperation have made the treatment of this form of heterophoria most gratifying. The latter element is essential. An enthusiastic and full coöperation is necessary. It is not possible in every instance to restore the muscle balance to normal, but success, as a rule, is in direct ratio to the energy and time which the patient is willing to devote to the purpose.

Refraction.—As in the other varieties of heterophoria, careful refraction under a cycloplegic is the first step in the treatment of the case. It would seem to be unnecessary to advise the student against the prescribing of glasses while the patient is under the influence of the cycloplegic. Unfortunately, it is a practise which is followed almost daily. Only after the effects of the cycloplegic are no longer operative, should an attempt be made either to prescribe glasses or to determine the muscle balance with the proposed correction placed in the trial frame. The single exception to this rule occurs in myopia. A full correction may be prescribed in myopia as found under the cycloplegic. Manifestly, however, such patients should be asked to return after the effect of the drops has disappeared, to determine the muscle balance and for further treatment if required. The reasons for a full myopic correction are patent. It is the rule in prescribing for moderate degrees of myopia, and it is doubly essential when an exophoria exists, to bring about a normal degree of accommodation for varying distances, in order also to restore a normal converging power. This is the "accommodative" type of exophoria. The exophoria may not disappear entirely after the wearing of the lenses, but in an average case after a week or ten days of constant use, a normal balance will be restored to the patient's entire comfort. If recovery is not complete, as shown by the symptoms and by the Maddox rod tests, appropriate treatment to be described under the head of "prism training" will complete the cure.

A hyperopic sphero-cylindrical error will require special consideration. Low hyperopic spheres with hyperopic astigmatism need no serious consideration at any age. Spheres under 0.75 diopters can be safely omitted, if the patient is not presbyopic. A full hyperopic spheric correction will tend to increase an existing exophoria, or to state it differently an under-correction will require greater accommodation on the part of the patient and will thereby stimulate convergence. If the full cylindrical correction is worn, an accommodation of 1 or 2 diopters will do no appreciable harm in a patient under thirty years of age. After thirty-five years, every diopter of spheric error must receive due consideration in relation to the amount of near work which the patient may be called upon to perform. The rule, therefore, to be followed in moderate degrees of hyperopia, 1 to 3 diopters, is to prescribe as much of the spheric error as the patient's near work will demand, bearing in mind the fact that an under-correction will aid the exophoria which may be present.

Hyperopia of high degrees, 4 to 9 diopters, require special consideration. High degrees of hyperopia, like myopia of moderate degree, lower central vision even early in life, and increasingly so with each decade after adolescence. It is manifestly necessary under these conditions to give first consideration to the requirements of central vision and second, consideration to the exophoria. The same rule, however, applies as in moderate degrees, namely a cutting down of the spheres which may safely be made in the interests of central vision, will aid in the correction of an existing exophoria. It is well also to call attention to the necessity for careful centering of the lenses in high degrees of hyperopia in order to prevent prismatic effects unless one does so designedly. Any residual exophoria which may call for treatment may be met as indicated under "prism training."

Exophoria between the ages of thirty-five and forty-five years must be met as individual cases demand. If an exophoria of 4 diopters or more are present, the demands of oncoming presbyopia may require two pair of glasses. Exophoria for distance may be controlled by a moderate but deliberate decrease in the strength of the spheres. A fuller correction for the near-point may be prescribed with suitable exercises to overcome the exophoria.

When presbyopia is established, plus lenses should be prescribed as indicated. Any annoying exophoria must be dealt with by suitable training.

Reading Distance. -Orthophoria for distance with a moderate degree of exophoria for the near-point which glasses do not correct, may be reduced to a minimum by instructing the patient to read and do near work at a maximum distance from the eyes. Exophoria diminishes as one recedes in fixation from the near-point toward infinity. On the other hand, certain occupations, such as mechanical drawing, require concentrated effort at close range. This increases the tendency to exophoria and may be met by prisms at rest, this to be discussed shortly.

Medical Treatment. Logically with the prescribing of glasses, a careful inquiry will be made as to the patient's habits of life, his occupation, the amount of exercise indulged in, his temperament, etc. Any measures which will tend to improve the general health, increase the general muscle tone, and stabilize an unbalanced nervous system, will help to hold in check a tendency to exophoria. To this end, hours of close work should be limited to a minimum; outdoor exercise should be encouraged and the patient should be as free from worry as is possible. Increasing doses of nux vomica have been advocated with good results in some cases. In others, the cutting off of tobacco and cigarettes have brought about happy results. Coffee and tea are more apt to aggravate an esophoria than to increase exophoria. Aside from the use of large doses of strychnia and nux vomica, the author has found no specific medication of value in the treatment, other than that which may be directed to underlying physical conditions referred to above.

Prisms Incorporated in Glasses for Constant Use. -Although comparatively little use has been made of prisms to be worn base out or in, there is a distinct but limited field for their use in exophoria. Reference has been made to the use of prisms placed bases in for binocular vision at close range. The ophthalmic surgeon after thirty-five years of age will find the addition of a 2 diopter sphere with a 2 diopter prism base in, an advantageous addition to his distance correction for close-up intra-ocular operations. Similar additions have been made to the distance correction of patients whose occupations require the minute inspection of stamps, coins, etc. The mechanical draftsman can be made more comfortable for work at close range with a 1 diopter sphere placed base in before both eyes, by means of hook fronts, this to be removed when work is resumed at the usual working distance.

In addition to these practical uses of prisms adjusted for exophoria at close range, there are instances when prisms may be similarly prescribed in *carefully selected* cases. If all methods of treatment fail to relieve a harrowing exophoria, and the patient will not consent to, or the physician deems it inadvisable to resort to operative interference, the patient may be made tolerably comfortable by prisms ground into his lenses or added to his correction by "hook fronts." The author does not recommend the indiscriminate use of prisms so placed for the correction of exophoria, but he can testify to a few instances in which patients have been made comfortable by such prescriptions. For this purpose, a 1 or 2 diopter prism is ground into the near segment, base in before each eye, or the same is incorporated in a "hook front" with the near correction. In a few instances, although the exophoria may be considerable for distance, 5 to 9 diopters, the patient may continue the fight for single binocular vision for distance, but abandons binocular vision for the near-point. In such cases, 1 or 2 diopter prisms placed in the distance correction may do much to relieve the patient's discomfort.

Although the author feels that many, like himself, find the limited use of prisms incorporated in the ametropic correction of great value in a number of cases, their indiscriminate use before thorough training has been practised, is to be discouraged. This use of prisms in exophoria should only be made after proper treatment has failed and operation is declined, or in those of advanced years in whom training does not offer much hope of success.

Special Exercises Other than Prism Training.—Many forms of ocular exercises are practised, all of which are alleged to be of value in increasing the converging power of the eyes. The most practical of these is the practice of approximating a pencil, for example, to the eyes as the point is intently fixed by the patient. When the point blurs and becomes double, it is again carried away from the eyes and gradually brought to the convergence near-point. This exercise may be of some value if the patient concentrates upon the exercise and notes carefully when the pencil breaks into two images.

With the same object in view, the author instructs patients to spend brief periods each day in reading aloud to himself in the following manner. A good-sized type is selected and reading is begun at arm's length, and the book or manuscript is gradually brought to the near-point of convergence, when the type becomes blurred and diplopia will follow or one eye only will fix and the other will diverge. When this point is reached, the book is again moved off and gradually, as before, is brought to the nearest point possible with clear binocular single vision. This form of exercise is more apt to hold the patient's attention because central or foveal vision alone is being used. Reading aloud will insure greater concentration than the simple pencil exercise to which reference has been made.

These exercises are not of great value in high degrees of exophoria. In low degrees, 4 or 5 diopters, when the amount of exophoria is the cause of symptoms, some good may be accomplished by their use.

Prism Training.—Prism training has been practised by many and passed into discard because the method practised failed to produce results. In some instances, failure may be attributed to a lack of coöperation on the part of the patient. More often, however, the cause of failure may be due to faulty methods and technic. The author's earlier experiences were of this character, but by persistence and renewed efforts, a method of training was evolved which has proven to be of great help to him and to many others who practise similar methods. As the minute details are essential to success, the method as practised will be given rather fully.

Before prism training is begun, the refractive error is carefully corrected, the muscle balance is taken for the far- and near-point, the convergence near-point is determined in centimeters and the lateral ductions are recorded.

Instructions are given in the office for the use of prisms at a distance of 6 meters or 20 feet from the muscle light. The patient's converging power determines the prism strength with which to begin. If the adducting power has been recorded as 15 diopters, for example, two 5 diopter prisms are placed in the patient's hands for use. Trial frames are not used. The prisms are square or rectagonal. The patient is instructed to hold a prism base out before the right eye and observe that two lights appear, but immediately fuse into one. It is essential that patients should recognize this fact to obviate mistakes in the future. If they recognize the fact that two lights appear momentarily but fuse, they grasp the object of the training. Prisms are placed base out, because the light is displaced thereby to the outside of the macula, and a converging movement must be executed in order to bring the maculæ into position to secure single vision.

After the first prism is overcome by fusion, with this prism still in place the second 5 diopter prism is added before the left eye and the fusion process is repeated and both prisms are held in place for a few moments.

In the second step, a 2 diopter prism is added to the 5 in the right hand and fusion is obtained. To this is added the 5 in the left hand, making a total of 12 diopters. In the third step, 7 diopters are added in the left hand. Additions are made step by step until the patient reaches his maximum for that period of exercise and the amount is recorded. In the example given above, it is not unusual to be able to add a total of 25 diopters at the first sitting. A muscle balance is now taken, and a decided improvement in the degree of exophoria, especially for the near-point, gives the patient the assurance that much may be accomplished by systematic training.

The patient is given minute instruction in each step taken, in order that he may carry out the real training at home. He is then given an order to one of the many optical houses, with whom one can arrange to rent sets of prisms suitable for this purpose. The sets should include two each of 2, 3, 5, 10, 15 and 20 diopters. He is also given the following type-written instruction slip to help him to conduct the exercises at home. One or two fifteen-minute periods are devoted daily to the training, always at 20 feet carefully measured.

SIMPLE RULES TO OBSERVE IN TRAINING WITH PRISMS IN EXOPHORIA.

1. Use a lighted candle placed at 20 feet if practising at distance, and at 2 feet if at the near-point. (A dark background is best.)
2. Hold the thick part (base) of the prisms out before the eyes.
3. Hold them accurately, in the horizontal position, and *do not tilt*.
4. Always begin with the prism before the right eye.
5. Looking at the fixation light, hold before the right eye the lowest degree prism in your set which will separate the light into two, probably 3 or 5 diopters. Endeavor to fuse these two lights into one. When you have done this, place before the left eye a lower degree prism than the one you are using before the right eye. This will cause the light to separate again. In the same manner as you fused the two lights before, now endeavor to bring the two lights together.
6. In building up, make the additions to the right eye mostly, 2 and 3 diopters at a time, until 30 or 40 diopters are reached, when the prisms may be divided more equally between the two eyes.
7. Endeavor to reach 75 diopters or more.
8. Practise for fifteen minutes daily with glasses on while exercising.

He is asked to report in one week for a check-up to correct errors in technic, and to determine the results of training, as to comfort and as to the state of the muscle balance. If the technic is thoroughly understood, return visits are requested in one or two weeks and at less frequent intervals thereafter as the case may require. The length of the training required, varies with the individual and with the degree of the exophoria. A minimum time should be four weeks but many will require eight weeks or longer.

The results, with few exceptions, are good. As a rule, the average patient will report comfort after one week of proper training. It is not always possible to bring about a normal balance in the higher degrees of exophoria. What is accomplished, even in those cases which show a residual exophoria for the near-point, is comfort and a reduction of the exophoria to within limits, which can be overcome by the fusion faculty without discomfort to the patient. What degrees of exophoria can be overcome by this method has often been asked? The author has been able to relieve an exophoria of 5 to 7 diopters for distance and corresponding 10 to 15 diopters for the near-point.

When the patient has reached satisfactory results, he is instructed to purchase two 15 and two 20 diopter prisms for home use. With these he is asked to practise at intervals of a few days to a week in order to "keep himself fit." He can, as a rule, determine for himself the state of his adducting power by the ease or difficulty with which he can fuse, after periods of rest, these higher prism denominations. There is a tendency in some to regress, but a brief period of training as a rule suffices to place them on their former basis. In others the results are permanent.

Quite a few find it difficult to learn the knack of fusion at first. It may not be possible for some of low adduction power to accomplish much at the first office visit. The tension, strange surroundings, and embarrassment at failing to succeed even with low prisms, occasionally make the first attempts almost futile. These patients are carefully instructed and are sent home to try the experiment in the quiet of their own homes, where they usually meet with success and subsequent attempts improve from week to week.

How far should patients be instructed to carry the experiments? An average of 70 diopters is usually sufficient, although patients of low adducting power at first may eventually be able to overcome 80 and 90 diopters.

Can the exercises be carried too far? This is possible in a few instances, and for this reason, return visits should be made to check-up upon the state of the muscle balance. When a patient shows orthophoria or little exophoria for distance, but a considerable degree for the near-point, the exercises may be limited to practise at a near-point of from 33 cm. to 1 meter from the light. This same rule is followed, if the exercises bring about 3 or more diopters of esophoria for distance. When, however, even 2 diopters of exophoria are present at 6 meters, the exercises should be practised at the usual 6 meter distance. To one who understands the underlying principles of heterophoria and fusion, it might not be necessary to add that it is immaterial as to whether the prisms are placed before one eye or both. The results must necessarily be the same. It is not a matter of one eye or the other doing the work. It is a question of fusion in which the eyes and the musculature share equally. When, however, a prism of high degree is placed before one eye, the image is so changed in form and color as to make it totally dissimilar to the light as seen by the other eye. This dissimilarity of the two images in shape and form and color makes fusion more difficult. When prisms of a high degree, therefore, are placed before the eyes, they should be so equally divided as to create images of similarity so as to aid the power of fusion and not hinder it.

Surgery in Exophoria.—The results obtained by prism training in even considerable degrees of exophoria are so satisfactory that the final step in the management of this form of heterophoria, namely surgical interference, rarely becomes necessary. In fact, the surgeon of large experience will study his case most carefully before he will advise surgical procedures. A patient of a highly neurotic temperament is not a good subject for surgery, whether the condition be one of esophoria or exophoria. The type of patient usually selected is one whose exophoria is of considerable degree for far and near, whose vocation demands many hours at the near-point, and whose condition is manifestly caused by some anatomical defect, such as referred to under etiology. In this type surgery may be of great value. Any surgical procedure which offers an increase of the converging power of the eyes is preferable to one which theoretically will diminish the diverging power. Tenotomy of the externi is not only valueless but dangerous. Recession of the externi is not accompanied by any risk, but the setting back of one or both external recti is not a logical procedure for the correction of this condition.

Resections, tuckings and advancements of one or both interni offer a choice of operations in which surgeons are guided by individual preference and by the degree of exophoria to be overcome. A correctly performed advancement in which muscle, capsule and conjunctiva are included, is the operation of choice. Preference is given to tucking of the interni by others.

Both operations have advantages and disadvantages. The surgeon of limited experience will probably encounter fewer serious difficulties in tuckings than in resections. At all events, if tucking is indicated in ophthalmic surgery, it is in the phorias (eso- and exophoria) in which the operation can be said to be of special value. The same operation is practised by many in the correction of moderate degrees of squint. In the lateral phorias, therefore, when only a moderate degree of latent deviation must be overcome, the operation may be practised to advantage. Resections with advancements are reserved for the correction of the higher deviations as found in squint. The combined operation rarely is indicated in exophoria.

As previously indicated, the relation of the prism duction power becomes a determining factor in the choice of operations. Even though weak convergence may be the rule, there are instances in which excessive divergence is a dominating factor. When such is the case, recession of one or both external recti may be indicated.

As a final word in the surgery for the correction of esophoria or exophoria, the author would remind the reader that although the various procedures would seem to be the same as when applied to the correction of concomitant squint, such is far from a true statement of fact. True enough it would seem that the difference is only one of degree. As a matter of fact, however, the results in the phorias are not as gratifying as in squint. In squint there is a definite amount of deviation which may be corrected by a properly performed operation. Parallelism of the visual axes enables the fusion faculty to again functionate as in a normal individual. In the lateral phorias, on the other hand, the underlying factor is one of innervation. In some instances, even after a considerable gain by operative interference, exophoria continues to manifest itself. The chief cause for failure in the author's experience is to be found in the fact that an operation cannot correct a faulty innervation which really is a function of the brain; nor can an operation upon a single muscle be expected to restore a normal balance when twelve extra-ocular muscles, as well as the intrinsic ocular group, are concerned in this act. It is, however, the only surgical means of approach. When all measures fail to overcome exophoria, operative measures, nevertheless, are justified, if the prognosis is guarded.

Following operation, prismatic and especially stereoscopic exercises, are again indicated if the best results are to be realized.

CYCLOPHORIA.

A tendency to a deviation of the vertical meridians of the corneæ from the normal, around their sagittal axes, is probably less understood and less discussed than any other organic or functional motor anomaly of the eyes. From a practical standpoint the subject is almost ignored. This is due in part to a general lack of knowledge of the phenomenon and in part also to the fact that many writers on the subject believe that cyclophoria rarely gives rise to symptoms. In fact, there is great diversity of opinion among authors. Some are in accord with Maddox[1] in his statement "in nearly all cases, non-paralytic cyclophoria causes no symptoms and requires no treatment." Others have been carried in their enthusiasm, like George T. Stevens[2] to believe that cyclophoria plays a dominant role in the production of all forms of functional motor disturbances of the eyes. Between these widely diverging beliefs, the careful student of the ocular muscles and of refraction will find a sufficient number of patients who complain of phenomena, which can only be caused by cyclophoria, to make a careful study of the subject worth while.

Nomenclature.—Cyclophoria[3] is the generally accepted term now used to express a tendency to a deviation of the vertical meridians of the corneæ from the normal. Plus cyclophoria indicates a tendency for the upper end of the meridian to lean templeward and minus cyclophoria signifies a tendency to nasal leaning. George T. Stevens introduced the terms plus and minus declinations for the same conditions and Maddox is credited with the introduction of plus and minus torsion. While all are correct, nothing is gained by the multiplication of terms. Cyclophoria harmonizes with the nomenclature now universally used for subdivisions of the generic term herterophoria. G. C. Savage[4] suggests a further subdivision into pseudo- and intrinsic cyclophoria. Pseudocyclophoria is used by Savage to indicate cyclophoria which results from the efforts of patients to overcome refractive errors and which, in a large measure, cease with the proper correction of such errors. Intrinsic cyclophoria is applied to deviation tendencies which result largely from anatomical and innervational influences. In order to harmonize with the subdivisions applied to other forms of heterophoria, the author will adhere to the terms *accommodative* and *essential* cyclophoria. In the former, the cyclophoria is associated primarily and fundamentally with ametropia and corrective accommodative efforts. In the latter group, "essential" expresses the basic character of the underlying cause.

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[1] The Ocular Muscles, 2d ed., p. 236.

[2] Motor Apparatus of the Eyes.

[3] The term cyclophoria, according to Savage, was proposed by George H. Price.

[4] Ophthalmic Myology.

Etiology.—In the section on Physiology, the author emphasized the fact that each muscle has primary and secondary, or subsidiary actions, the lateral rotators excepted when the eyes are in primary position. The internal and external recti are lateral rotators. The superior and inferior recti are concerned primarily with elevation and depression. In right and left rotation, in elevation and depression, these muscles are the leaders in their several groups. To the obliques is assigned the function of torsion or wheel motion, or of maintaining the vertical meridians in equilibrium. Regardless of the efficiency of the obliques in elevation and depression of the eyes, this primary or basic function of wheel motion must be thoroughly grasped in order to understand the manner in which cyclophoria may be produced. The superior obliques are intortors and the inferior obliques are extortors. When the superior group under act, extorsion is the result; if the inferior obliques are weak, intorsion is apt to follow.

There are instances in which it is difficult to determine whether the causative factors are wholly of the accommodative variety or whether faulty responses to innervations or anatomical abnormalities are totally or in part responsible. In many instances, probably all may contribute.

An example of the accommodative variety is found in oblique astigmatism. When a patient, suffering from oblique astigmatism of considerable degree, looks at a vertical or horizontal line, the image formed on the retina will lean toward the maximum corneal meridian. Unless the astigmatism is corrected by a cylinder, the corrective action of an oblique will necessarily be called into play to overcome the leaning. In uncorrected oblique astigmatism, as Maddox says, "corrective torsion becomes a life habit." This explains the great amount of headache and the nervous upset which so often accompanies astigmatism at oblique axes. It also explains the reason for latent cyclophoria when a cylinder is improperly placed with regard to the correct axis. With the proper correction, however, of oblique astigmatism all symptoms should disappear.

The effect of oblique astigmatism in the production of latent torsion is more easily understood by following the diagrams suggested by Maddox, but modified by the author to conform to anatomical methods of observation.

If a strong plus cylinder, + 4 *D*, is placed before the right eye at axis 135 degrees, and the subject is asked to look at the cross *A* in Fig. 61, the left eye being closed, the cross is observed as in *B*, the lines inclined toward the meridian of greatest curvature. The retinal image leans towards the meridian of the maximum corneal curvature. If the cylinder is placed before the left eye at axis 45 degrees, the cross will seem to be as in *C*. *D* in Fig. 63 represents the lines as they would be seen with both eyes if no attempt at fusion were possible. As this is impossible, concentration of both eyes upon

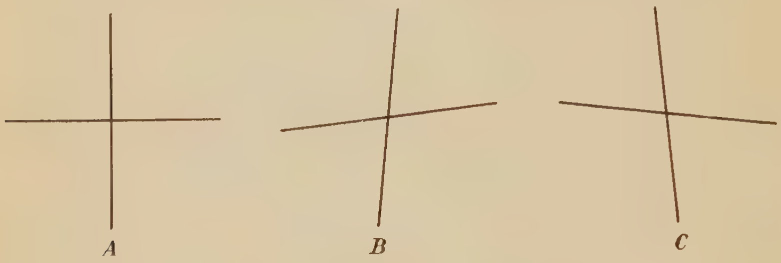

Fig. 61

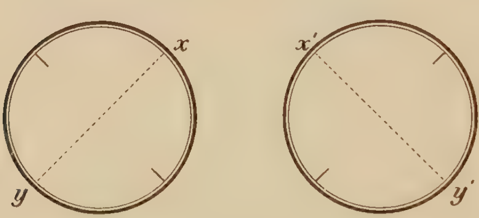

Fig. 62

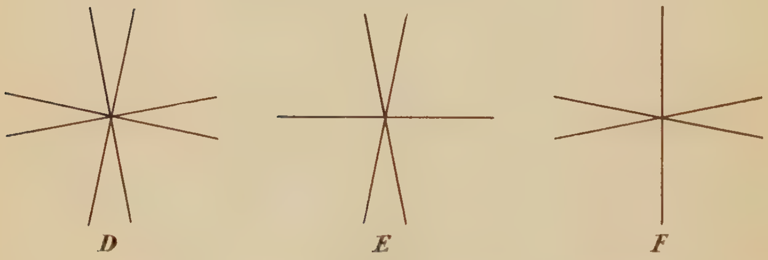

Fig. 63

Figs. 61, 62 and 63.—Diagrams illustrating the effect of a + 4D cylinder, placed before the eyes at oblique axes.

the horizontal results in perfect fusion as in *E* in Fig. 63, and if the vertical line is fixed, *F* is the result. The artificial oblique astigmatism produced by a plus cylinder, illustrates what happens when oblique astigmatism exists as a natural refractive error. This form of accommodative cyclophoria is correctable by proper refraction and the obliques are relieved of the necessity of constant over-action.

Another phase of cyclophoria is observed in near work, especially in the lower field. Convergence of the eyes in the lower field, as in reading, is brought about by the inferior recti, the internal recti, and in part by the superior obliques. With the downward pull of the inferior recti, there also is a coincidental tendency to extorsion which must be offset by the intorting action of the superior obliques. If extorsion is not properly balanced by the action of the superior obliques, plus cyclophoria may be the result. This is largely an accommodative type, although intrinsic or essential factors may aid in bringing it about, as for example insufficiency of the obliques.

The purely essential cyclophoria is due either to innervational disturbances, or to some form of anatomical abnormality In an individual of an unstable nervous system, failure of the obliques to respond normally to an innervation is as easily understood as failure of the lateral rotators to make a normal response. In such instances, there may be no anatomical abnormalities. The over-action or under-action of the obliques are simply an expression of an over-action or under-action of the ocular muscles in general.

Although Stevens was inclined to believe that insufficiency or over-action of the obliques, as observed in cyclophoria, was the underlying cause of other forms of heterophoria, clinical experience leads one to believe that the cause and effect relation is in fact reversed. For example, the complex act required to overcome an annoying hyperphoria, necessitates the combined corrective action of the vertical recti and the obliques. That cyclophoria should follow this effort is more easily understood than the reverse phenomenon, namely the production of hyperphoria because of an existing cyclophoria.

Cyclophoria may be unilateral or bilateral. It may be plus in one eye and minus in the other, it may be plus in both, or minus in both. A cause of cyclophoria, particularly of the unilateral type, may be some form of motor anomaly. This may be due to an improper insertion, or other defect, to some abnormality of the capsule of Tenon, or to paresis of an oblique.

Symptoms.—The symptoms produced by cyclophoria are probably of more consequence than is general conceded. They form a part, no doubt, of the general group of symptoms credited to other forms of muscular asthenopia, and rarely are separated. A specific instance, however, of symptoms due to cyclophoria is observed in oblique astigmatism. The severe headache and nervous upset, even nausea and vomiting so frequently noted in oblique astigmatism, is due in great measure to the corrective action of the obliques necessary to overcome the tendency to the leaning of the images.

After correct refraction and an apparently normal muscle balance, patients occasionally return with continued evidence of ocular discomfort. It is not unlikely that a careful study of such cases will uncover the fact that some at least may be suffering from cyclophoria. Stevens found an amplitude of 11 degrees of extorsion and slightly less for intorsion. It is generally believed that considerable degrees of either plus or minus cyclophoria can be overcome by the average individual without symptoms. In others, just as in hyperphoria, even moderate degrees of leaning may produce symptoms.

Head tilting, which is observed in ocular muscle palsies and in hyperphoria, is also a symptom of cyclophoria. In ocular palsies the tilting of the face is of greater value than head leaning to the shoulder in bringing the images to a common level. In the author's experience the so-called "shoulder tipping" is as much in the interests of correcting the leaning of the image as of lowering or raising the false image. In all cases of head posing, therefore, in which the muscle balance is normal, careful study should be made as to the presence of cyclophoria, either unilateral or bilateral when the leaning of the images is in the same direction, either to the right or left.

Methods of Studying Cyclophoria.—Many cyclophorometers have been devised for the detection and measurement of cyclophoria. They are for the most part adapted to laboratory use, and few have found their way into private office equipment. Price apparently was the first to design such an instrument which consisted of glass rods mounted vertically before the eyes with a double prism placed before one. The principles involved in this test have been incorporated in the modern clinical tests which are usually employed in the detection of this condition. The clinoscope of Stevens (Figs. 64 and 65) is probably the most practical and the one generally in use for laboratory study.[1]

For practical office use two Maddox rods may be carefully mounted or attached to a phorometer, if accurately placed by means of a spirit level, so as to insure that the rods are strictly vertical. The lines may be separated by a suitable prism so as to prevent fusion. A single rod may be used without a displacing

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[1] The interested reader is referred to Stevens (Motor Apparatus of the Eye) for full account of this instrument and its operation.

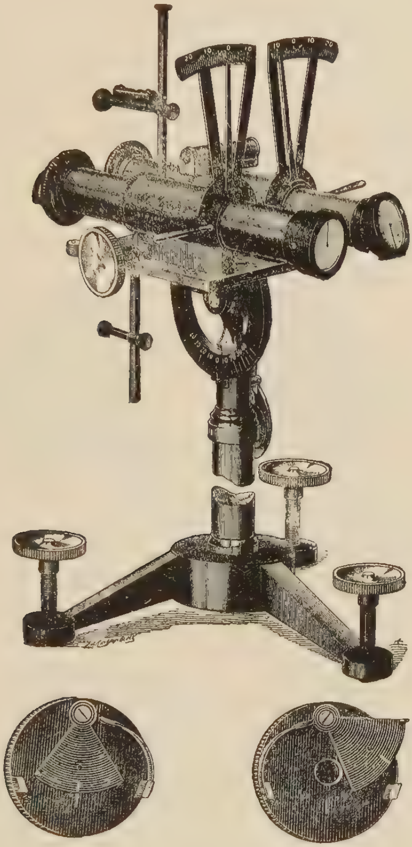

FIG. 64.—The clinoscope. (From Stevens' "Motor Apparatus of the Eyes.)

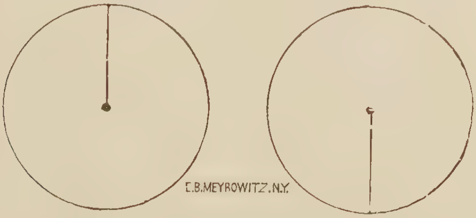

FIG. 65.—Objective lines for the clinoscope. (From Stevens' "Motor Apparatus of the Eyes.")

prism, if a plumb-line is dropped through the light, with which the line of light from the rod may be compared. Either of these methods will definitely disclose a plus or minus cyclophoria (Fig. 66). For the near-point a Maddox double prism may be placed before one eye and a line on a card in the horizontal may be focussed at 33 cm. The eye before which the Maddox double prism is placed will see two parallel lines. Between these lines will appear the line as seen by the other eye. If the three lines are parallel and horizontal there is no cyclophoria. A dipping of the middle line to the left will indicate minus cyclophoria of the left eye if the prism is before the right eye. If the middle image dips to the right the condition is one of plus cyclophoria (Fig. 67).

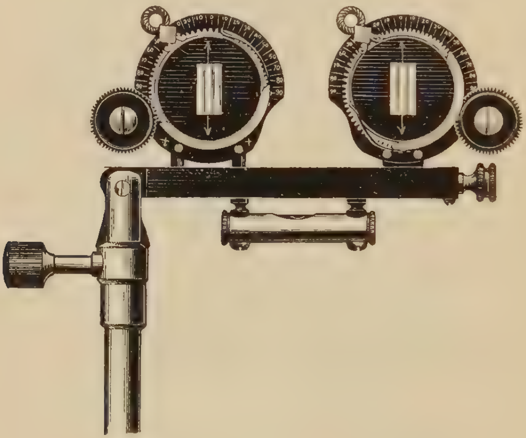

Fig. 66.—Savage's cyclophorometer. (Thorington.)

The false image, if inclined to the left, will indicate that the vertical corneal meridian is intorted because of insufficiency of the left inferior oblique. If this middle line dips to the right the left eye is extorted because of insufficiency of the left superior oblique. The position of the false image with reference to the vertical and horizontal meridians of the cornea is opposite to the leaning of the cornea. When the upper end of the corneal meridian leans to the temporal side the false image in the vertical will lean to the nasal side, and the horizontal false image will dip to the right. The diagrams in Fig. 67, *A* and *B*, are recorded by the anatomical method. The reader sees the lines, as it were, through a transparency.

When the double prism is placed before the left eye the image of the right eye may dip to the right or left. If the inferior oblique is weak the corneal and retinal meridian will lean to the nasal side the horizontal meridian will dip to the left and the resulting image will incline to the right—minus cyclophoria. If the superior oblique is weak the vertical corneal and retinal meridian will lean to the temporal side—plus cyclophoria. The horizontal meridian will dip to the right and the horizontal image will incline to the left (Fig. 68, *C* and *D*).

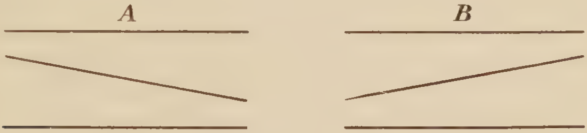

Fig. 67.—*A*, minus cyclophoria of left eye; the double prism before the right. *B*, plus cyclophoria of left eye; the double prism before the right.

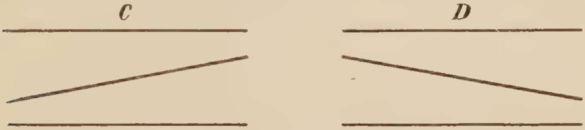

Fig. 68.—*C*, minus cyclophoria of right eye; double prism on left eye. *D*, plus cyclophoria of right eye; double prism on left eye.

The reader will observe that plus cyclophoria refers to the outward leaning of the vertical retinal meridian, and not to the leaning of the image. The latter is directly opposite. For example, in plus cyclophoria of the right eye the vertical *retinal meridian* incline to the temporal side; the vertical image will lean to the nasal side and the horizontal image will dip to the left. This harmonizes with the behavior of the image in hyperphoria. Right hyperphoria refers to the fact that the axis of the right eye is high and not to the image which is low.

In applying the test for the near-point, it is evident that the parallel lines in the eye before which the double prism is placed may lean instead of the central line which belongs to the fellow eye. It will be less confusing to the observer if the double prism is alternately placed before each eye, and the plus or minus cyclophoria is estimated with reference to the middle line in each instance.

The amount of leaning in the average patient is not considerable. If, however, the patient complains of ocular fatigue, which cannot be explained by ametropia or other forms of heterophoria, due consideration should be given to even moderate degrees of cyclophoria.

Treatment.—As previously stated, the entire subject of cyclophoria is not as well understood as other forms of heterophoria. The treatment of the same is no exception to the rule. The student will encounter no difficulty in the management of the accommodative variety. Correct refraction is the *sine qua non* in this group, as it is in all forms of heterophoria. A careful study of the patient's needs in the correction of the total astigmatism and accurate placement of the axes is the first essential. If wholly accommodative, a proper refractive correction should relieve all symptoms. If cyclophoria continues, such leaning of the images and persistence of symptoms must be attributed to "essential" factors. At this point the difficulties in treatment begin.

Influence of Hyperphoria.—The second step in the management of cyclophoria is careful correction of hyperphoria. Inasmuch as these cases are an unusual group, the state of the muscle balance should be determined after prolonged occlusion of one eye, so as to insure perfect disassociation of the two eyes when the muscle balance is determined. It is the author's belief that in these cases practically the entire minimum amount of hyperphoria so uncovered should be corrected by suitable prisms. By the "minimum" is meant the minimum level either for distance or for the near-point, when the hyperphoria is unequal. A proper ametropic and a full hyperphoric correction will reduce to a minimum the number of cyclophorics needing further attention.

Exercise of the Obliques.—In selecting methods of increasing the dynamic power of ocular muscle action, those which can be practised daily by the patient in his own home promise the best results. Unless such exercises can be applied with regularity, one cannot hope to obtain relief. Training, therefore, which is conducted wholly in the surgeon's office, is apt to result in failure, even though the method *per se* is an ideal one. The best results will be obtained by a combination of both types of training, home exercises and office methods which are carried out under the direct supervision of the oculist.

The *clinoscope* of Stevens is well adapted for office use, but largely impracticable because few offices are equipped with this instrument. Diameter disks are employed and the disks are rotated in such a manner as to require the action of either the superior or or inferior obliques as the individual case may require.

The cyclophorometer (Fig. 66), as devised by Savage, is more practical. It is operated in a manner similar to that of the clinoscope. As a substitute for the cyclophorometer, an ordinary phorometer may be used if it is provided with a spirit level and two Maddox rods accurately placed in the lens cells. They should be mounted vertically. A displacing prism is not employed. The two lines are fused and the rod before the cyclophoric eye is gradually rotated out or in as required after the method of measuring plus or minus cycloduction. Savage advises a rotation of not more than one-half the plus or minus cycloducting power of the eye. The rod is moved alternately in the direction which will exercise the muscle and back to zero or position of rest. For exercising the superior obliques the rods should be moved in the lower temporal arcs. Moving them in the lower nasal arcs in turn tends to strengthen the inferior obliques. The exercises should be continued for periods of ten minutes daily, but should never be pushed to the point of fatigue.

Utilizing the same principle, the author has experimented satisfactorily with two movable circles on a stereoscopic card placed in the stereoscope. By a rotating screw placed on the back of each circle, the vertical diameter of one circle may be rotated against that of the other. The device has the advantage of enabling the patient to practise daily at home.

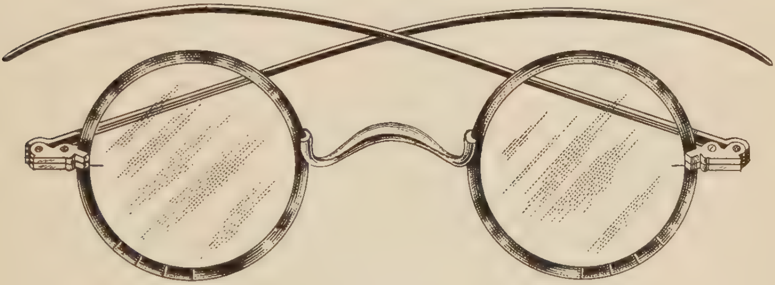

FIG. 69.—Savage's method of cylinder exercise in cyclophoria.

Exercise Cylinders.—Exercise cylinders, as recommended by Savage, may be used by the patient at home with good results. Cylinders ranging from +0.50 D to +1.50 D are placed in a specially constructed frame, as shown in Fig. 69, for weakness of the superior obliques. The marks on the rims are 15 degrees apart—from 90 to 45 degrees in the right and from 90 to 135 degrees in the left. The lenses are revolved in the frames to notch No. 2, and a horizontal line, 8 to 10 feet distant, is fixed for three seconds. The same line is alternately fixed with and without the lenses for three-second periods, the entire exercise continuing for about five minutes.

The lenses are then moved to the third notch and the exercises are continued.

If the inferior obliques are weak the lenses are rotated similarly as above, but in the direction of the lower nasal quadrants. Care should be observed, according to Savage, to work longest at the first and second notches, and only for short periods when the third or maximum exercise notch is reached.

The same author has recommended the use of weak *rest cylinders*, +0.50 D. For insufficiency of the superior obliques, plus cyclophoria, the cylinder is placed between 90 and 135 degrees in the right and between 90 and 45 degrees in the left. These cylinders were used by Savage in non-astigmatic cases. In astigmatic patients the cylinder is moved in a similar direction, but to a moderate extent, about 5 degrees in cylinders of about 1 diopter. In either instance this author claims to have given much relief to his patients. The fallacy of the method lies in the fact that such displacement tends to blur the images, and after all, is but a "rest" for the obliques. Nothing is gained in the way of adding strength to the muscles. As Savage adds, "It is better to make the muscles strong by exercise or else correct the cyclophoria by operating on one or more of the recti." This summary by Savage harmonizes with the convictions of others who have made an extended study of cyclophoria. In fact, in all but the "accommodative group," due for the most part to oblique astigmatism, it is more than likely that much time and inconvenience to the patient can be saved by proceeding to the more satisfactory surgical measures if the cyclophoric symptoms are pronounced and demand treatment.

Operative Treatment.—The recti lend themselves more readily to surgical intervention than do the obliques. In fact, inaccessibility of the obliques is a good surgical reason for a "let alone" policy, insofar as these muscles are concerned. Because of the precision with which a change in the position of the eyeball may be affected by altering the attachments of the recti, surgery is logically limited to this group. Savage was disposed to confine his surgery to the vertical recti; Stevens preferred a change in the attachments of the external and internal recti.

Plus cyclophoria, insufficiency of the superior oblique, may be corrected by a tenotomy of the nasal fibers of the superior rectus or by advancing the nasal side of the inferior rectus. Stevens secured the same results by advancing the upper border and receding the inferior border of the internal rectus. In choosing between the two methods, the surgeon should give consideration to the fact that in changing the attachment of the vertical recti, the normal action of the muscle is increased, or diminished. Furthermore, it is logical to readjust the ratio of power between two synergistic muscles, when one is under-acting. Shifting the attachment of the internal rectus adds an additional function to a muscle which normally is only an internal rotator. In uncomplicated cyclophoria the preference should be given to the vertical recti.

Minus cyclophoria is corrected by a division of the temporal fibers of the superior rectus or by an advancement of the temporal fibers of the inferior rectus. Similarly the lower border of the internal rectus may be advanced and the upper fibers set further back.

The presence of hyperphoria, esophoria, or exophoria, with cyclophoria will have a determining influence in the choice of operation. A coëxisting hyperphoria with plus cyclophoria would be favorably influenced by tenotomy of the inner border of the superior rectus. If esophoria or exophoria is a complicating condition surgery should be directed to the internal or external rectus. Before undertaking any surgical measure the operator will do well to weigh the evidence carefully and select a constructive procedure. To this end he may elect to do an advancement of the outer fibers of the superior rectus instead of tenotomy of the nasal fibers.

In the rare instances in which there is a coexisting double hyperphoria or a double hypophoria, according to Savage, one may favorably influence these conditions by partial tenotomies of the superior recti in the former and the inferior recti in the latter. Whether the cyclophoria is plus or minus will determine which border of the recti shall be tenotomized or advanced.

PART III.

HETEROTROPIA OR CONCOMITANT SQUINT[1]

In keeping with the nomenclature employed in the sections of Part II, heterotropia is the generic term under which will be included all forms of concomitant squint, in contradistinction to paralytic squint. Squint and strabismus are synonymous terms, more frequently employed than that of heterotropia, and they will be used in this text for greater simplification.

BINOCULAR VISION.

The extensive over-lapping of the visual fields in man implies also binocular vision of a high degree. In order that this may be accomplished, it is essential first that corresponding retinal points shall be so placed that impressions carried to the brain shall be the same, and the two impressions, therefore, will be seen as one. Second, a fusion faculty becomes a necessity, without which single binocular vision is difficult to understand. Absence of either of these important functions of the eyes need not necessarily result in manifest squint, but single binocular vision implies that both are present. When fusion is absent or defective the conjugate action of the ocular muscles may be so carefully preserved that the eyes may remain in parallelism. More often manifest squint makes its appearance. On the other hand, as observed in heterophoria, if the fusion faculty is strong and elastic, although corresponding retinal points may not be perfectly coördinated or placed so as to receive exactly the same impression, single binocular vision may nevertheless be maintained. Confirmation of this fact may be found in placing in a stereoscope the diagrams *A* and *B* of Fig. 70.

Although many factors may enter into the causation of squint, the state of the fusion faculty is an important determining element.

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[1] In order to avoid the necessity of reduplication, it will be assumed that the reader will refresh his memory on the anatomy and physiology of the ocular muscles in Part I, before beginning the study of concomitant squint.

No better classification of the degree of fusion has been given than that of Claude Worth:[1] (1) Simultaneous macular perception; (2) true fusion with some amplitude; (3) a sense of perspective. In the first group images exactly alike may be so superimposed that no sense of diplopia will be present, but there will be no amplitude. In this group each eye can perceive at the same time the images which are placed before them, but there may be no effort made to fuse them into one. For example, *F* and *L* will be recognized as the objects placed in the stereoscope, but no attempt will be made to fuse the two letters with the formation of *E*. In the second group there is true fusion with some elasticity. The *F* and *L* can be fused into *E*. Amplitude is shown by the ability of the patient to change the angle of the tubes to a limited extent and still maintain the *E*. In the third grade a sense of depth is added to fusion.

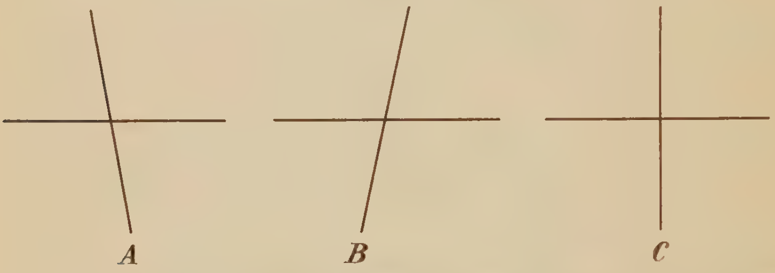

FIG. 70.—The horizontal lines will be fused and seen in *C* as in *A* and *B*. The vertical lines, although oblique, will be psychically fused and seen as in *C*.

The third degree of fusion is demonstrated by the ability of the patient to fuse images in which the principle of Mr. Berry is incorporated. The inner circles in Fig. 85 if displaced toward one another produce the sense of relief; if displaced away from one another or toward the outer rims of the large circles, a sense of depth is obtained.[2] The patient can tell, for example, whether a tub is placed top up or bottom up. In the first group the fusion faculty is wholly absent or so undeveloped that it rarely becomes possible to develop it into a true fusion faculty. Those, however, who are grouped in the second grade, showing true fusion with some amplitude, as a rule can be trained by the amblyoscope or stereoscope to develop a sense of depth and relief. The importance of this classification will be appreciated as the etiology of the various forms of concomitant squint is developed, and its even greater importance will force itself upon the student in the discussion of treatment.

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[1] Squint, 4th. ed., p. 12.

[2] This change in the position of the inner circles can be effected in the amblyoscope by shifting the cards from one tube to the other. In the stereoscope, Wells' charts, in which this principle is incorporated, may be employed.

DEVELOPMENT OF THE FUSION FACULTY.

The fusion faculty, fusion sense, or fusion center, as it is variously called, if present at all, asserts itself early in an infant's life. As pointed out by Worth, the fixation reflex, macular vision, and a fairly normal vertical muscular balance are present at birth. Shortly after birth the infant can fix for a moment upon a bright light if the room is fairly dark. Either eye may fix, or accidentally both may seem to be attracted by the light for the moment, but the eyes will soon wander. The faculty grows in strength and the eyes for the most part are in parallelism during the first year. Even in a normal child, however, there may be frequent lapses when the eyes may be observed to deviate inward and occasionally outward. These lapses are observed during attacks of crying from slight pain, as in colic, or when the infant is below *par* in health. They are normal lapses to which the term "occasional" squint may be applied. These occasional or periodic attacks of squint should be clearly differentiated from "incipient squint," a form which may also be designated as "premonitory squint," in which the same eye frequently is observed to turn in or out.

At the end of a year the fusion faculty is fairly well established in the average normal child. Just when fusion reaches its highest degree of development is not definitely known. It is generally agreed, however, that a defective fusion faculty may be trained up to the seventh year (Worth). The author has been able to train weak fusion in patients as old as eighteen years. A schoolboy, aged eighteen years, now under the author's care, has had his fusion restored to normal after operation, by daily exercises with the stereoscope.[1] It is not unusual to find children of twelve to fourteen years of age respond promptly to similar treatment after successfully performed advancements and resections. The fact remains, however, that one, as a rule, meets with more successes in training a defective fusion faculty if the training is instituted before the seventh year.

At this point it is well to state definitely a matter which does not seem to be thoroughly grasped by all in the profession. *The stereoscope is of no value in training fusion in squint, unless the degree of deviation is very slight.* If there is a manifest squint present the stereoscope should be replaced by the amblyoscope or the Priestley Smith fusion tubes in order that the stereoscopic pictures can be placed within the reach of the fusion faculty. This can be done by inclining the tubes to correspond to the angle of squint.[1] After operation, if the eyes are brought to parallelism, the stereoscope may be used. The stereoscope can only be of service when the fusion breadth is equal to or greater than the diplopia breadth.

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[1] A patient, aged forty-six years, in which fusion was restored by stereoscopic training after operation, is reported by Dr. Otis Wolfe (Am. Jour. Ophthal., vol. **10**, 51).

ESOTROPIA OR CONVERGENT SQUINT.

The several types of concomitant squint to be studied have in common many points of interest in etiology, methods of study and correction. Each, however, will be better understood by an individual study rather than by a collective one.

Esotropia is manifested by the turning in of the visual axis of one eye. While one eye fixes upon an object, the other, or squinting eye, is directed inward, and in so doing crosses the visual line of the fixing eye at a point between the object of fixation and the eye. This is not the only sign or symptom of the condition, but the one which is apparent and which gives character to the form of squint under discussion. Associated with it, there may be a defective or absent fusion faculty, a refractive error of varying degree and character, suppression of the image of the squinting eye, and amblyopia, congenital or acquired as the result of disuse. The squint may be constantly present in the same eye, *monocular squint,* or alternating in type, *alternating squint.* Furthermore, whether monocular or alternating, the degree of squint represents the deviation of both eyes manifested in the convergent eye. A final characteristic is the angle of deviation, which remains practically the same as the eyes are rotated from extreme right to the extreme left. The notable exception to this rule will be referred to under the discussion of "fields of rotation." It is this characteristic of esotropia which so readily distinguishes it from paralytic squint, in which the angle of deviation increases as the eyes are rotated toward the field of action of the paralyzed muscle. A second exception occurs when fixation is suddenly carried from a distant point to a near-point. In this instance the squinting eye may tend to turn in excessively because of the necessary relation between accommodation and convergence.

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[1] If the angle of squint is approximately corrected by *loose prisms* placed in the stereoscope this instrument may be substituted for the amblyoscope.

Classification of Esotropia.—The clinical types of convergent squint are: (1) Occasional; (2) incipient or premonitory; (3) constant or established squint.

Occasional esotropia, called by Worth *spurious squint* of infants, is the squint which may be observed in normal infants during the first three or four months of life. It may be observed when the child is disturbed by pain from gastric or other disorders. It is fleeting in character and does not deserve special consideration.

Incipient, or *premonitory squint,* occurs at the same period of life or throughout the first year. It is more frequent in its recurrence, persists for more than a moment and often is found to recur in the same eye. When convergence of this character is observed after the child is three months of age, the patient should be under constant observation until one is definitely sure that it is a real forerunner of either type to be considered. As a rule, if the onset of permanent squint is sudden, following one of the precipitating factors which will be discussed, the incipient or premonitory character of the squint is definitely determined. If it proves to be of the occasional type, all traces of deviation disappear with the development of the fusion faculty.

Permanent or *established squint* is either monocular or alternating.

Monocular Esotropia.—Because of the fact that squint always represents the deviation of both eyes, but is apparent only in one, objections have been raised to the use of the term monocular squint. It is, however, so satisfying in expressing the type of squint in contrast to the alternating type, that there seems to be no good reason why the term should be discarded. "Constant squint," a term occasionally used, does not express the condition because most squints are constant. It is, however, expressive of the condition as opposed to alternating. The term monocular will, therefore, be used in this treatise. The reader will understand that the term implies nothing more than that the same eye invariably squints, while the other eye constantly maintains fixation. When the cover test is applied and the fixing eye is occluded, the squinting eye, if not amblyopic, will fix. When the cover is now removed the squinting eye may continue to hold fixation for an appreciable time. More often, the squinting eye will turn to its original position and the constantly fixing eye will again fix. In the first instance the monocular character of the squint can be determined by observing the patient closely. Preference will usually be shown by the patient to fix uniformly with the same eye. If any doubt exists it usually can be determined by studying the refractive error and the state of the fusion faculty. As a rule, the patient will fix with the eye which possesses the better vision. If the fusion faculty is present, but undeveloped, the form of squint usually found is of the monocular type. If the fusion faculty is totally absent, and visual acuity is about the same in each eye, the type of squint, as a rule, is alternating.

Alternating Squint.—In the alternating type the patient is able to use either eye and, as a rule, does not give preference to either one. It is a form of squint which is apt to begin in the first or second year. The refractive error is often negligible, rarely of a high degree, and anisometropia is not in evidence. Visual acuity is about the same in each eye, or if a refractive error is present the vision is correctable to the same degree of efficiency. An important characteristic of this form of convergent esotropia is absence of a fusion faculty. One notes from time to time in the literature that fusion has been developed by assiduous practice on the part of the patient. It is entirely within the range of possibilities that a defective fusion faculty may be present in true alternating squint, and if present it may be developed. In the author's experience this rarely occurs.

Occasionally a squint which began as an alternating type finally becomes a monocular one—the patient for some reason electing consciously or subconsciously to fix at all times with the same eye. If this transition takes place in late childhood or early adolescence amblyopia, which is so apt to occur in the early monocular types, does not develop.

Excepting under the condition just mentioned, or in the event of a macular lesion, which may occur *de novo*, amblyopia is not present in alternating type. It is but natural to expect that this should be so, because the constant use of either eye at will or automatically does not permit of the deterioration of macular vision.

MONOCULAR ESOTROPIA.

Monocular and alternating types of squint are so definite in their special etiology, characteristics and treatment, that they will be discussed separately.

Etiology. A fusion faculty is always present but, as a rule, undeveloped. The author does not feel that it is necessary to defend his position in assuming that the state of the fusion faculty is the important etiological factor in concomitant squint. Those who doubt that this position is tenable are respectfully referred to Worth's excellent treatise on *Squint* (fourth edition, p. 59). If a careful study of squint and its relation to the state of the fusion faculty is not convincing, Worth has set forth the reason for his belief in definite language, which should be read with care and applied to one's every-day experiences.

In monocular squint, if amblyopia is not of such a degree as to prevent its demonstration, a fusion faculty is found present in practically all cases. It may be quite defective, but patience in the use of the amblyoscope usually is rewarded by the finding of Worth's second degree of fusion, even though it is rudimentary. In the author's experience, if present, it can be developed.

Refractive Errors. The refractive error is of much importance. The error, as a rule, is rather high. In the earlier years of a child's life this in itself may not be sufficient to bring about squint if the fusion faculty is of average development. When, however, the child begins to spend much time at close range, in school or in the ordinary plays and entertainment common to children of four or five years, the increased accommodation necessary at the near-point, in the presence of 3 or 4 diopters of hypermetropia, tends to bring about an increased convergence. If the fusion faculty is normally developed this tendency to excessive convergence may be overcome, but if the fusion faculty is not strong the hypermetropia may be the active factor in precipitating squint. Most children are born hyperopic. In view of this fact, one must assume that something other than hypermetropia is present in those who develop squint, to distinguish them from the thousands having equally high errors who do not. This theory, spoken of as Donder's theory, or the accommodative theory, based on the relation of convergence to accommodation, might seem to be sufficient in itself in certain cases to explain squint. Notably are those cases which clear up and recover single binocular vision after the hypermetropic error is corrected by suitable glasses at a sufficiently early period. The author has on his records a number of these cases. One is that of Mrs. M., who has a convergent squint of about 20 degrees. When her glasses are worn a correction of:

O. D. + 0.25 S = +2.75 C ax 90 = 6/5
O. S. + 0.25 S = +0.25 C ax 90 = 6/5

the squint entirely disappears only to return whenever the glasses are removed. Her fusion faculty is good. In this instance the squint must be explained by the relation of convergence to accommodation. These cases, however, are isolated and few when compared with the large number of cases which one sees in a busy practice. In many instances, with high hyperopic errors, the angle of squint is lessened by the wearing of glasses, but the total squint is not corrected. In fact, in a majority of these cases even training of the fusion faculty will not restore the eyes to parallelism. The reason is evident. The images are beyond the breadth of fusion. Correcting the hypermetropia reduces the squint to the extent of the meter angle of convergence commensurate with the degree of hypermetropia corrected, but the fusion faculty cannot fuse the images because they are beyond the reach of fusion. If the squint in these cases is corrected to within reach of the fusion faculty by means of an operation, the fusion faculty completes the cure. In other instances the hypermetropic error is insufficient to be even active in the production of squint, or the error may be one of myopia, but squint nevertheless appears. The student will do well to regard the refractive error as an important factor in many cases, and perhaps the dominant factor in some, but he should study each case in its relation to a defective fusion faculty. In a majority of instances the squint is the direct result of a defective fusion faculty, accompanied by a high hyperopic error.

Anisometropia.—Anisometropia, or an unequal refractive error, is frequently observed in unilateral esotropia. It is not difficult to understand why the better seeing eye should become the fixing eye, when vision is reduced in one by a higher degree of hyperopia. The fact remains, however, that anisometropia is not of unusual occurrence, and that only a limited number of these patients become squinters. The accommodative theory cannot explain this fact. When one reviews his case histories, and observes the large number of cases of unequal refractive errors on his books, with normal or nearly normal muscle balance, he is forced to the conclusion that the fusion faculty in those who squint, with this refractive condition present, is not sufficiently strong to hold the eyes in parallelism and squint follows.

The relation of anisometropia to amblyopia exanopsia is easily comprehended. If vision in one eye is good, and high hypermetropia renders the other insensitive, amblyopia becomes assured unless a normal fusion faculty can hold the two eyes in fixation. Anisometropia, therefore, should be regarded as an important factor in the precipitation of esotropia if the fusion faculty is not properly developed. It should also be regarded as a cause of the monocular type of squint, and as a large contributing factor in the development and amblyopia from disuse.

Muscle Theory of Squint.—The so-called muscle theory of squint scarcely needs consideration. Originally the theory attracted many adherents. From the number of tenotomies which were performed in the past, and the number which the author regrets to say are performed even today to correct convergent esotropia, there may be a greater number of advocates even now than we are willing to concede. It is true that muscle abnormalities, undeveloped muscles, abnormal attachments and origins, and muscle (external recti) injuries in childbirth and otherwise, may occasionally be found as a factor in the formation of squint. All but those due to injury are exceedingly rare and enter into comparatively few cases as an etiological factor. They should, however, be regarded as occasional causes.

Injury to the external rectus during childbirth is of common occurrence. Gifford[1] has devoted many pages to the discussion of paralysis and paresis of the external rectus in its relation to childbirth injuries. This condition, however, should not be regarded as a cause of concomitant esotropia, but of paralytic squint. One of our modern text-books on the ocular muscles includes "congenital palsies" of the external rectus and "obstetric injuries" in the etiology of concomitant esotropia. This inclusion manifestly is in error, as both conditions are instances of paralytic esotropia.

Some of the older writers regarded the interni as unduly short in convergent squint. The fact that squint may disappear entirely during sleep or profound anesthesia is sufficient evidence that the theory is untenable. We would accord to muscular anomalies a very small place in the etiology of any form of concomitant squint.

Defective Outward Rotation.—This, so often observed, is regarded by some as an indication that the external rectus is defective or weak. This apparent weakness should not be interpreted as a cause, but as an effect of a long-standing squint. It is an indication that some anatomical changes have taken place in the muscles and the capsule of Tenon, and full external rotation is not possible because of these changes. Worth found the degree of limitation directly in proportion to the duration of the squint.

Heredity.—A third important factor in the etiology of esotropia is that of heredity. The squint itself is probably not bequeathed directly from parent to child, but no one can doubt that refractive errors are so handed down. It is also probable that the same may be said of the fusion faculty. It is not unusual, therefore, to observe esotropia in a parent and in one or more of the children. Such a group of three children was under the author's care recently, the mother also suffering from the same condition. Worth found hereditary influences in approximately 52 per cent of a series of 711 cases studied from this standpoint.

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[1] Am. Jour. Ophthal., February, 1926.

Precipitating Causes.—While the three great factors, namely, a defective fusion faculty, refractive errors and heredity, might be regarded as the predisposing causes of unilateral esotropia, there are other causes to be considered. These causes are of less importance to the physician, but have a certain precipitating influence which the layman stresses as the real cause of the condition. These factors are: (1) Profound mental shock or disturbance occasioned by sudden fright or fear; many parents have given as the cause "frightened by a dog," great fear occasioned by the sudden appearance of anything which might frighten a child in its tender years. (2) Children are often reported to come out of a convulsive seizure with manifest squint, which has remained and become permanent. (3) Squint has been reported to follow any of the diseases of childhood, specific or contagious fevers, and especially measles and whooping-cough. Needless to say, any of these contributing factors may play a role in the presence of one or all of the predisposing causes. Their action as precipitators should be carefully differentiated from the real predisposing causes. Without a defect in the fusion faculty, it is difficult to conceive of any one of the so-called contributing causes as sufficient to precipitate permanent squint. These contributing causes, however, are more frequently observed in monolateral squint than in the true alternating type. The latter often dates from birth or shortly after, whereas the monolateral type is more apt to appear between the second and fourth years of childhood.

Amblyopia in Esotropia.—The subject of amblyopia is more fully discussed on page 182 under Symptoms of Esotropia. At this point it is sufficient to state that congenital amblyopia is rarely observed in squint. When macular pathology is evident with the ophthalmoscope, such pathology explains the existence of squint. The cause and effect relation is easily determined. In the more obscure cases in which there are no gross evidences of macular disturbance, it may be difficult to determine whether an existing amblyopia is congenital or acquired. A careful study, however, of central vision and of the central fields will help to make the differentiation.

Symptoms. The most conspicuous symptom in monolateral esotropia is the deviation of one eye. In this variety of squint one notes that the same eye always turns in while the other eye always is used in fixation. This is well exemplified by the cover test. The test should be made at 6 meters. If the cover is suddenly removed from the good eye, the squinting eye fixing, the non-squinting eye immediately moves into position to fix. Occasionally if the refractive error is moderate and amblyopia is absent, the squinting eye may remain in fixation temporarily. This does not, however, signify that one is dealing with an alternating type. The habitually fixing eye very soon returns to fixation, and only an effort on the part of the patient, during the cover test, enables the squinting eye to hold fixation temporarily. A little observation by the physician will usually determine the fact that the patient invariably gives preference to one eye.

One should also observe the behavior of the squinting eye at the near-point, or during dynamic convergence. This is best observed by asking the patient to fix on a near object directly in line with the visual line when looking at infinity. During this act no movement will be observed in the fixing eye, but the squinting eye will be observed to turn in during the act of dynamic convergence. The increased convergence will represent the dynamic convergence of both eyes at this point.

Ordinarily the angle of squint is regarded as constantly the same. In a measure, this is a point of differentiation from paralytic squint. It can be graphically observed in the dark room. If one throws a light from an ophthalmoscope upon the cornea at a definite distance, say of 1 meter, first from the right side, then from directly in front, and finally from the left of the patient, having the patient constantly fix his eye on the ophthalmoscope, the corneal reflexes will be observed in the same relative position on the corneæ from each of the three points of view. This argues for constancy. If, however, the angle of squint is observed when the patient fixes at 6 meters and again at the near-point, a chance will be observed due to dynamic convergence.

There is another exception which may be noted in most cases. This variation is due to different states of health. When the child is well there is little, if any, variation in the angle of squint, but most children will tend to show some increase in the squint when ill. These variations are not constant nor are they uniformly the same. They are innervational disturbances to which one does not attach any special significance.

Occasionally one sees a case of another type. Changes in the angle of squint occur frequently. At times the deviation is slight and almost absent. At other times, convergence of a high degree is observed. These children have unbalanced nervous systems. A similar analogous phenomenon is observed in a type of esophoria which has been described. Both conditions are difficult to correct. Stereoscopic training after operation in the esotropic variety does a great deal of good, as it also is the best form of training to relieve symptoms in esophoria.

Growing Out of Squint.—In the management of squint it so often happens that the child is brought for treatment only when it is referred from school with a request that the eyes be properly cared for. The usual excuses for failure to bring the child earlier are two: Parents either declare the family physician advised against any treatment until the child is of school age, or they were advised to wait as the child may *grow out of the squint.* Aside from the fact that success in the management of squint depends largely upon the early institution of treatment, it is pertinent to ask "Does the child grow out of a squint?" In the author's experience, such a possibility is rare in cases of well-established strabismus. Squint of moderate degree has been known to disappear about the age of puberty in rare instances. In such cases the probabilities are that a fairly well-established fusion faculty saves the situation. It is alleged that the development of the orbit at this age may assist in the curative process. The author has never been able to give any credit to this probability. At all events, *the spontaneous cure of esotropia is of so rare an occurrence that it should never be held out to the parents as even a remote possibility.* On the contrary, delay in the adoption of corrective measures is the greatest obstacle in the way of perfect cures.

Amblyopia Exanopsia.—Amblyopia is of frequent occurrence in monocular esotropia. In fact, it is largely confined to this form of concomitant squint. It is not present in the alternating type, and but rarely in exotropia. It is, therefore, quite in order to discuss it at this time.

If one excludes those cases in which definite macular pathology is evident, congenital amblyopia can be said to be rare. In the author's experience, a few squinters have been observed with congenital amblyopia. These instances were in patients in which the history at least indicated squint of too recent origin to have acquired amblyopia after the appearance of the squint. As the degree of amblyopia was moderate, it was assumed that in all probability the amblyopia was congenital rather than acquired. One, however, has no assurance that such is the case.

Worth found few cases of congenital amblyopia, with visual acuity lower than 6/12 and in his group he found none having vision lower than 6/60. In the acquired form, on the other hand, central vision falls much lower, and few, if untreated, are observed with visual acuity of more than 6/15. Furthermore, in amblyopia exanopsia, retinal sensitivity is disturbed for a considerable distance beyond the macular region. Worth believes that the blind area in some instances extends 15 to 20 degrees around the point of fixation, an area in which only light sense may be present. The extent of this relative scotoma is largely responsible for the difficulty which one encounters in some instances in obtaining fixation. While profound central visual loss is directly responsible for lost fixation, there are instances in which the patient can scarcely make the attempt to bring the squinting eye to fix. In these instances the extent of the scotoma is an important factor. In the author's experience the blind areas in amblyopia exanopsia do not extend more than from 5 to 7 degrees toward the nasal side of the point of fixation. Above and below the point of fixation the amblyopic area may be greater than to the nasal side. The scotoma reaches its greatest limits toward the blind spot of Mariotte, and in a few instances is continuous with the normal blind spot. If the central scotoma is not continuous with the normal blind spot the latter, in practically all cases, is absolutely and relatively enlarged. In the field studies submitted (Figs. 71 to 74) the extent of the scotoma is more or less directly related to the duration of the amblyopia. The same may be said of central vision.

The few cases in which the scotoma is caeco-central, including the blind spot and central fixation, raise an interesting question as to the nature of amblyopia exanopsia. The following facts are evident: (1) The amblyopia extends beyond the papillo-macular bundle; (2) the scotoma reaches its greatest extent in the direction of the blind spot; (3) it is a relative area, indistinct for white and never definitely absolute even when central vision is very low (light sense is preserved); (4) it has been shown in recent studies that treatment improves even the most profound cases, although the degree of improvement depends upon the duration of the scotoma, the age of the patient and the degree of central visual acuity; (5) peripheral fields in the amblyopic eye are smaller than the fields of the fixing eye.

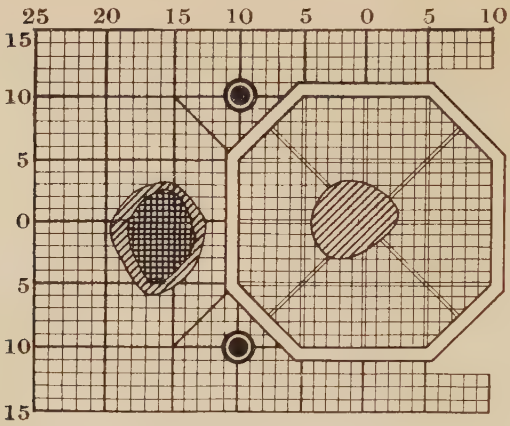

Fig. 71

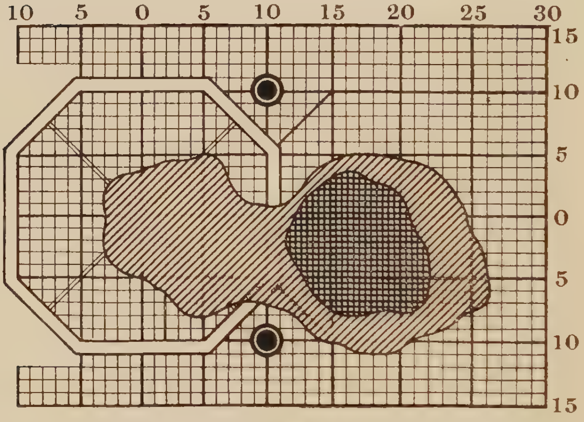

Fig. 72

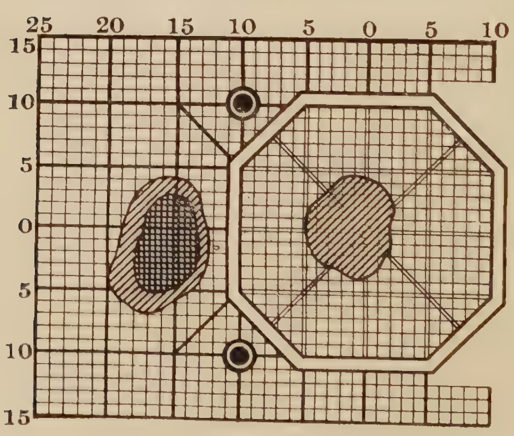

Fig. 73

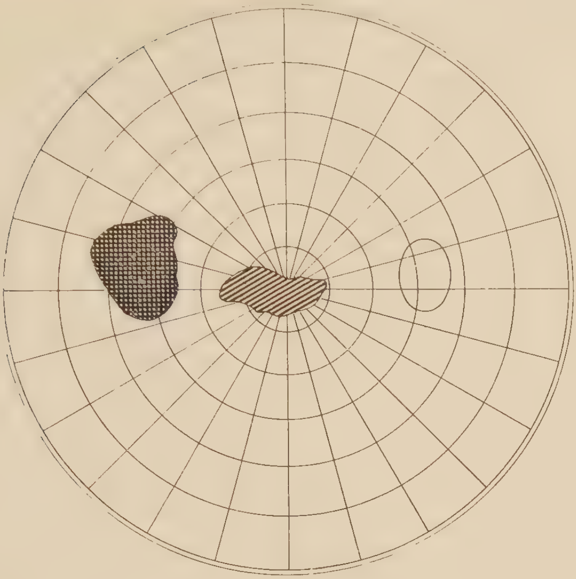

FIG. 74

FIGS. 71 to 74.—Types of central field defects in amblyopia exanopsia.

In view of the studies which are as yet incomplete, the author does not feel definitely committed to the exact character of the scotoma, but the above facts seem to confirm the existing belief that the scotoma is the result of disuse. It is caused by the squint and not the cause of the deviation, as claimed by some.

Aside from the monolateral character of the squint in which amblyopia exanopsia is found, the phenomenon is especially apt to develop in two types of esotropia. The first type is accompanied by anisometropia and the second by high degrees of hyperopia. Either condition tends to a rapid development of profound amblyopia. If the refractive error is negligible or of moderate degree, even though the squint is unilateral, amblyopia is less likely to develop.

The question naturally arises, To what extent can either form of amblyopia be corrected by suitable training? Little, if any, improvement is observed from energetic training in congenital amblyopia. In amblyopia exanopsia much may be accomplished. This phase of treatment will be thoroughly discussed in the section devoted to treatment. It is pertinent to add at this time that recovery or even improvement depends upon: (1) The degree of visual loss; (2) the age at which treatment is begun; (3) the absence of organic macular pathology, even though it may not be visible by means of the ophthalmoscope. Failure to improve the vision of an amblyopic eye in young children up to seven or eight years of age, in the author's experience, arouses a suspicion of organic macular disturbance as the cause of the amblyopia and a factor in the production of the squint. Furthermore, in the rational treatment of squint two conditions decide the question as to whether single binocular vision may be restored or cosmetic results alone may be obtained. These conditions are: (1) The presence or absence of a fusion faculty; and (2) the presence or absence of amblyopia. These two barriers form an *impasse* which make single binocular vision impossible. The student, therefore, will do well to give his closest attention especially to the latter. In the author's experience, amblyopia exanopsia is the barrier which is seen most frequently as a cause of failure. At the same time, it is largely preventable and correctable and, therefore, calls for special consideration.

False Macula.—As the angle of squint is rather uniformly maintained, the extra-macular point in the retina of the squinting eye, which receives the same impressions as the macula of the fixing eye, may become more than usually active and sensitive by constant use. It can manifestly not become more acutely sensitive than the rod and cone elements with which it is supplied and hence cannot develop vision of much acuity. Its constant activity may, however, render it more alert and after operation there may be a phantom shadow which occasionally annoys patients. This is fleeting in character, and need not be considered in operative interference. While the squint lasts it cannot be said to be a corresponding retinal point with the macula of the opposite eye. The two points are not so connected as to register the same image in the cortical cells. Its insensitivity does not make it a competing factor with the true macula of either eye. A more correct view of the so-called "false macula" is, it is a purely psychic phenomenon.

Suppression of the Image in the Squinting Eye.—Characteristic of any form of concomitant squint is absence of diplopia. In this it is unlike the annoying symptom of diplopia encountered in paralytic squint. Suppression of the images of the squinting eye is the explanation. This applies to macular vision as well as to peripheral. The patient will rarely complain of diplopia. It may be brought into existance by placing a red glass before either eye. A difference in the color of the images may enable the patient to see both. In certain patients, however, it is difficult to arouse diplopia even by this method.

Although the patient has automatically learned to suppress the image of the squinting eye, one should not assume that the eye is to all intents blind. It must be recalled that in the binocular field of vision each eye has a zone of vision to the temporal side which finds no counterpart in the other eye. As these patients avoid accidents as others whose eyes are straight, there is a certain amount of activity in this zone at least which enters into the visual act.

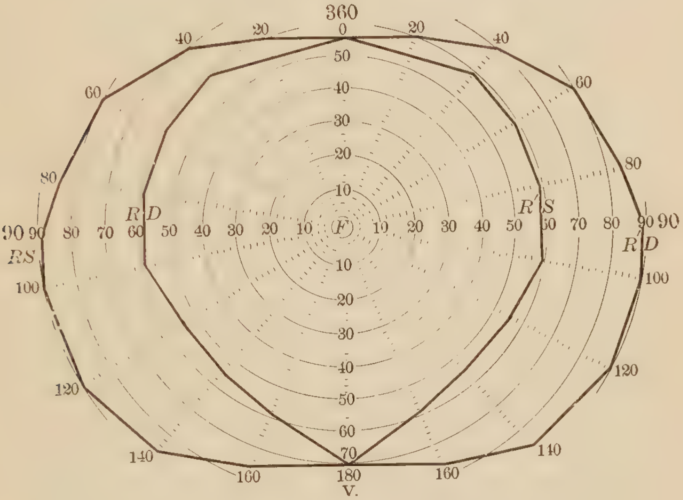

FIG. 75.—Binocular fields. (From Wilbrand and Saenger, after Förster.)

The zones *RS–RD* and *R′S–R′D* (Fig. 75) are independent zones, not having corresponding areas in the fellow eye. In these areas one would not look for diplopia for the reason given. In the large zone, *R D–R′S*, every point should have a corresponding retinal point in the fellow eye. Fusion, however, is concerned only with a comparatively small area, as shown in Fig. 76—an area approximately 3 degrees in the vertical and laterally about 12 degrees. This is the true fusion area, according to Savage, or at least the fusion area which is constantly in use and, therefore, active. In the balance of the binocular fields, because of physiological diplopia, retinal suppression is so usual that the average patient readily adjusts himself to the altered relations brought about by the deviation of the squint.

Suppression of vision is sufficient to prevent interference, but one cannot assume that the retina of this eye is totally inactive. The avoidance of accidents and the ability to catch moving objects to the side of the squinting eye is evidence of retinal activity, dissociated but in some way properly correlated with the dominant vision of the other eye.

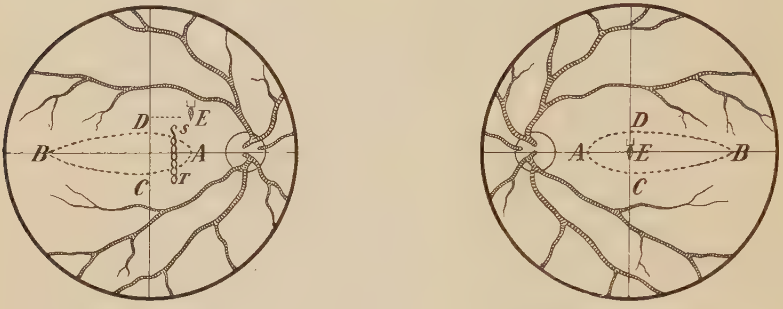

Fig. 76.—Fields of binocular fusion, *A D B C* as suggested by Savage.

Lost Fixation.—In high degrees of acquired amblyopia *of long standing* the patient may at least in part lose the power of fixation. This is in direct relation to the degree and the duration of visual loss. As previously pointed out, the author believes that the extent of the amblyopic area is also an important factor. If the squint remains uncorrected no improvement can be expected. It is possible, however, by the methods employed to correct the amblyopia, to restore fixation to a remarkable degree. This phase will be discussed fully in the section on Treatment.

Limitation in External Rotation.—In esotropia of long standing there occasionally is limitation in external rotation. This fact is possibly one of the reasons which led to belief in the muscular theory of the origin of squint. It should be observed that this limitation is only observed in cases of long standing. The reasons for the same are to be found in the anatomical changes which must necessarily develop in the muscles, capsule and check ligaments of the squinting eye. Similar but more marked muscle and capsule changes are observed in paralytic squint of long standing. There is this difference, however, that marked contractures develop in the opponent of the paralyzed muscle, and these contractions result in permanent organic cellular and fibrillar changes of a high degree. As a means of differentiation, the fields of rotation serve a useful purpose. In paralytic squint of the external rectus there is a distinct indentation at the center of the temporal contraction in the horizontal meridian. The obliques, being intact, are capable of a little more temporal pull than is the external rectus. The temporal rotation, as shown in the diagram Fig. 77, will, therefore, differ from that of weak temporal rotation of esotropia, as shown in Fig. 78.

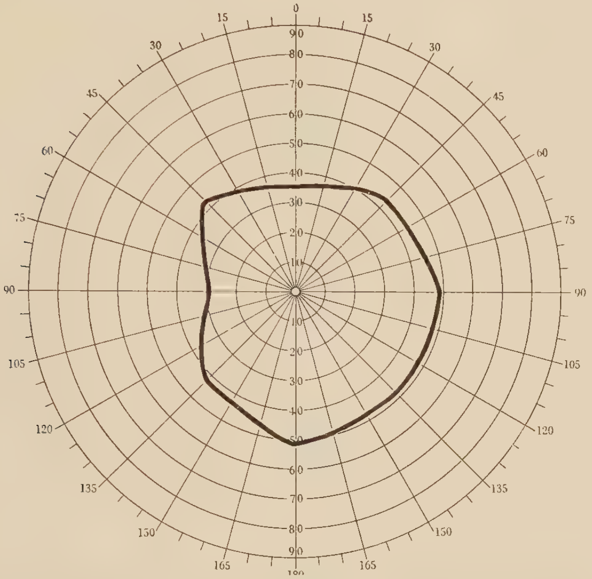

Fig. 77.—Field of rotation in paresis of the right external rectus.

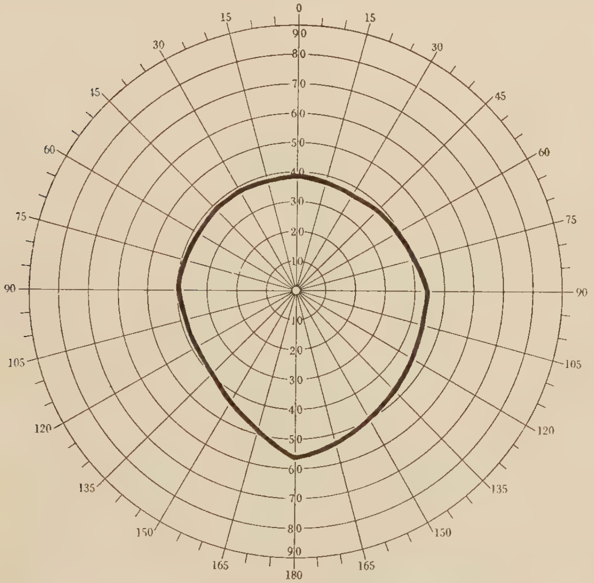

Fig. 78.—Field of rotation in monocular esotropia of the right eye.

Studies to be Made in Monocular Esotropia.—One cannot hope to obtain satisfactory results in the management of monocular squint without a thorough and exhaustive study. It is the type of squint most frequently encountered and, if properly managed, yields a higher percentage of cures than any other form.

History of the Case.—The age of the patient when squint first appeared should be carefully determined. Was it gradual or sudden in onset? Was it preceded by a period of premonitory squint, or did it follow any of the precipitating factors mentioned? Is it constant in character or are there periods of variation? These are some of the questions which should be asked.

Heredity.—In studying the history for hereditary tendencies, it is of interest not only to determine the number of cases in the family, but when possible a knowledge of the dominating refractive error. Does myopia or hyperopia predominate?

Treatment.—Treatment, if any, which the patient has received is of much importance. In the early treatment of esotropia correction of the refractive error and training to prevent or correct acquired amblyopia are the two phases which claim our attention. What, therefore, has been done in this direction is of the utmost importance.

Study of Central Vision.—Central vision in each eye should be carefully determined at the earliest possible moment. In children under school age, from two and a half to six years, Reber's test chart is the most satisfactory. Although the figures on this chart do not all conform to the requirements of the Snellen test type, based on a five-minute angle, it is sufficiently accurate to furnish satisfactory acuity values. In the case of younger children who are shy and unfamiliar with the objects on the chart, the author instructs the parents to purchase one of these charts for home use. The pictures are cut into squares and by kindergarten methods the child is taught the names of each at home. After a few weeks of this study the author has found even young children will give very satisfactory answers. To determine whether amblyopia is present, one may resort to Worth's ivory balls. In lieu of them, white marbles of varying size may be substituted or even bits of squares of white paper may be dropped on the floor. With the fixing eye occluded, the child is asked to pick up the bits, while the physician observes whether the child can accurately locate the bits of paper. If the child is of school age the regular Snellen test chart should be used.

One notes in many hospital records such notations for vision as "too young," "illiterate," "vision not obtainable." The state of vision is the first important point to be determined unless one is dealing with an infant in arms. Every means should be exhausted to learn, if possible, whether amblyopia has developed.

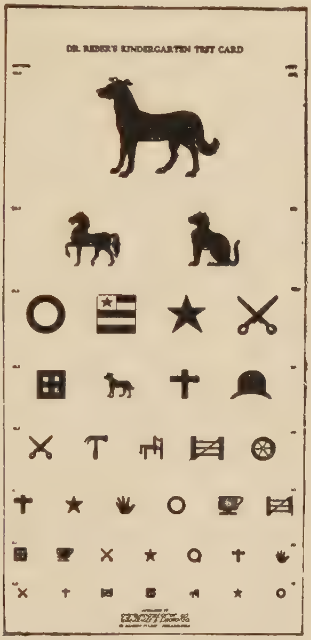

FIG. 79.—Reber's object or kindergarten test-card. (Courtesy of Dr. Howard F. Hansell and P. Blakistons' Sons Co.)

Power of Fixation.—Ability to fix is determined by the cover test if the child is old enough to engage in attention. If too young, a ready means of determining the power of fixation is that of throwing the light from an ophthalmoscope into the eye, each eye being alternately occluded. The bright light usually insures the attention of the child, if fixation is possible.

Character of the Esotropia.—By the same measures one can observe whether the esotropia is unilateral or alternating in character. In the cover test the right eye, for example, should be covered and uncovered while the child fixes at a point 6 meters distant. This will determine the child's ability to fix with the right eye. The same test is now carried to the left eye in the same manner. If the patient continues to fix with either eye for a period as both are uncovered, the condition is probably one of alternating esotropia, but not conclusively so. Further tests and observations should be made to learn whether the child habitually gives preference to one eye. In this, the statements of the parents and the careful scrutiny of the surgeon, while the child is under observation, will assist in arriving at the proper conclusion. Although the child may fix for a period with either eye, following the cover test, its habitual behavior will determine whether it is a unilateral or alternating squint.

If during the cover test the same eye always returns to fixation as both eyes are uncovered, the case is one of the monocular type. In young children the test by an ophthalmoscopic mirror will, in most instances, classify the esotropia.

Paralytic or Concomitant Esotropia.—In this differentiation the history of onset should be of first consideration.

Second, limitation of movement, as the eyes are engaged in attention, and the fixation object is moved from side to side, is, as a rule, definite and differentiating. In paralytic squint there is limitation of movement when the paralytic eye is carried in the direction of the paralyzed muscle. If the squint is concomitant, there is, as a rule, full lateral excursion as indicated by the corneæ reaching the outer and inner canthi. If any doubt exists the dark-room experiment is more refined. Light is thrown upon each cornea by means of an ophthalmoscopic mirror, first from the right side and then from the left of the patient, engaging his attention. If either eye fails to respond fully, a change in the angle of squint will be noted, as indicated by the position of the corneal reflex with reference to the pupillary center.

Angle Kappa.—As a rule, the presence or absence of the angle kappa is ignored. A positive angle kappa, the visual line to the inner side of the pupillary center in monocular fixation, tends to seemingly decrease the angle of the esotropia. A negative kappa, the visual line to the outside of the pupillary center, tends to increase an existing esotropia. As the angle kappa rarely amounts to more than 5 degrees, and usually less, its presence or absence is not of great importance in high degrees of squint. In low degrees, however, its significance increases in importance and its presence should be carefully noted. In older children and in adults its value when present should be carefully measured by the method described on page 49. The student should be cautioned lest he may mistake a positive kappa for a moderate degree of esotropia. The cover test, if carefully applied, will always differentiate the two conditions. If esotropia is not present there should be no movement under cover. Any movement under cover will indicate a manifest squint or a lateral heterophoria.

Monocular Rotations or Field of Fixation.—In all children old enough to respond and in all adults the field of fixation should be carefully plotted. The method has been described on page 73. Each eye should be studied separately, the other eye being occluded and the eye under observation being carefully centered on the perimeter. A letter of large size should be selected as a test object. The patient should be instructed to follow the letter with his macula to the extreme limits on the arm of the perimeter and the points where clear vision ceases should be carefully noted and recorded by the anatomical method either on a perimetric chart, as in Fig. 37, or on the diagram (Fig. 36).

No difficulty will be encountered in making the record by the anatomical method, if the operator will face the patient with the back of the chart held toward the patient, and due regard is given to the *patient's* right and left.

Measurement of the Angle of Squint.—One naturally will make a rough estimate of the probable angle of the squint by simple observation.

The Hirschberg method is a little more accurate, but not entirely satisfying. As a preliminary measure, in estimating the angle, it commends itself because it requires little time. The light from a luminous ophthalmoscope is brought close to the eyes. As the patient fixes the light the surgeon observes the position of the corneal reflex on the non-fixing eye, with relation to the margin of the pupil. If the reflex is noted at the outer edge of the pupil the degree of convergence is about 15 degrees. When the reflex is situated midway between the pupillary margin and the peripheral border, the degree of squint is from 30 to 35 degrees. When the reflex is observed at the limbus the angle of squint is approximately 45 degrees. It will be observed that the squint plus the degree of dynamic convergence are measured by this test.

Priestley Smith Tape Method.—Measurements may be made by means of the arc or tangent. In the latter group are included Worth's deviometer, Maddox's tangent scale, and Priestley Smith's tape. The former are useful for a public clinic, or a large private office if a multiplication of instruments is not objectionable. The Priestley Smith tape is simple, easily constructed, and fairly accurate. It consists of two tapes joined by a brass ring large enough to slip over a finger. Each tape should be 60 cm. in length from the center of the ring. A nurse holds the end of one tape to the side of the child's orbit while the operator slips his ophthalmoscope handle through the ring on the other end of the tape. The light from the ophthalmoscope is reflected on to the corneæ, and the reflexes are carefully observed on the corneal surfaces. The second tape is now held between the surgeon's thumb and first finger, and the child is asked to follow the finger as it is moved out until the corneal reflex falls in the center of the pupil of the squinting eye. As the tape held by the surgeon is graduated in centimeters, the tangential readings are carefully observed by direct reading. At a radius of 60 cm., each centimeter corresponds approximately to 1 degree. This method is sufficiently accurate for clinical work. It has the added advantage that a child of almost any age can be measured with a little patience. If the tape cannot be purchased from local dealers, it is easily constructed. The marking of the tape in centimeters will not be difficult. If the tape is made by the surgeon, it is desirable to mark the measuring arm in degrees of tangential value. The value of each degree desired on the tape may be obtained in millimeters by multiplying the natural tangent of the degree in question by 600 mm., the radial distance of the ophthalmoscopic mirror from the eye expressed in millimeters. The quotient will be tangential measurement expressed in millimeters.

Arc Measurement.—Worth objects to the perimeter because of alleged difficulty in technic and because it does not take into consideration the "angle gamma." In this treatise, *angle kappa* is retained as the logical term for the angle which is encountered clinically. To this latter objection one must add that practically all methods which utilize the light reflex on the cornea, are open to the same objection. The "angle kappa" should be observed in all cases and should be measured by the perimetric method and added in esotropia if positive, or substracted if negative. In the author's routine daily work, the perimetric method is always employed for children who are old enough. A child, aged three years, can be measured without difficulty. Its accuracy commends it. In many of the studies here set down, the surgeon may encounter difficulties in the very young. It is assumed, however, that in infancy one must forego many investigations which are very valuable, until such age as will admit of accurate answers.

For these measurements, either the old type of perimeter or the Ferree-Rand instrument may be used. The patient is placed with the chin supported so that the squinting eye will be opposite the point of fixation. The patient is now instructed to look not at the fixation point on the perimeter, but at an object 6 meters distant. The light from a luminous ophthalmoscope is now passed along the arm of the perimeter until its light is reflected in the center of the pupil of the squinting eye and the degree of deviation is read on the arm of the perimeter.

On the Ferree-Rand perimeter, Ferree recommends that the fixing eye be centered on the presbyopic device arranged for infinity. The angle is then measured as indicated above. From a table of tangents, the tangent for the angle of squint measured is determined and to it is added 0.003 times the interpupillary distance in millimeters. The sum will represent the corrected tangent of the angle of the squint. This angle may be found by noting the angle which corresponds to the tangential sum thus obtained. Although this is laboratory technic, of a high order, it is enclosed for those who are interested in the more scientific phases of the subject.

Prismatic Method.—In a limited number of cases with fairly good visual acuity, one can measure the angle of squint by means of prisms and a Maddox rod. The Maddox rod is placed before the fixing eye and prisms are added base out until the line of light passes through the light. This is an accurate method, but applicable to but a limited number of cases for obvious reasons. In its application, the angle kappa can be ignored.

Refractive Error.—This study is properly included under Treatment. In the preliminary study, however, one should roughly determine the gross refractive error which is present. For a preliminary survey, such measurements should be made by means of the ophthalmoscope. A more detailed study will be given in its proper place.

Condition of the Fusion Faculty.—A study of all the phases of esotropia thus far discussed would be incomplete without a proper classification of the case with reference to the condition of the fusion faculty. In very young children, under three years, little can be done to obtain a knowledge of the fusion faculty. Until the child is old enough to use the amblyoscope, one must mark time in the passing of final judgment as to just what may be expected in the way of a permanent cure. Some children are responsive as early as the third year, whereas others are difficult to handle until they are five or six years of age. In determining the degree of fusion, if present, or its total absence, the amblyoscope must be converted into a game into which most children will enter if they are approached in the proper manner. The cards to be selected first are a rat and a trap, a bird and a cage, etc., by means of which one can determine the presence of simultaneous macular perception. The second stage or that of fusion can be studied by means of an *F* and an *L* if the child is old enough. If this can be converted into an *E*, even though the third degree cannot be elicited, the prospects of good results from fusion training are very encouraging. If the *F* and *L* cannot be used, Fig. 83, or any similar chart may be substituted to determine the presence of fusion.

It is of the utmost importance that the operator should satisfy himself that not only the first of these tests can be successfully carried out, but that the second is also possible. If the first only

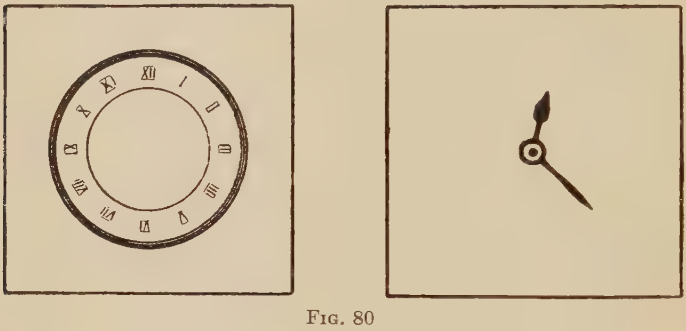

FIG. 80

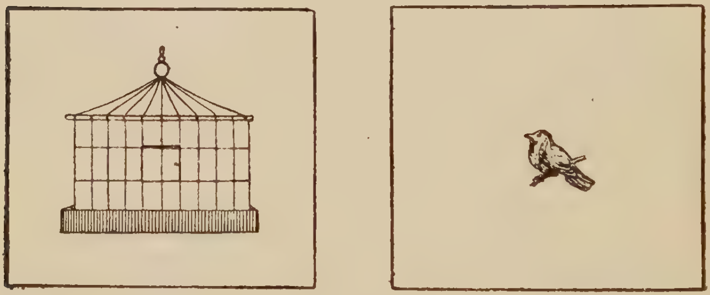

FIG. 81

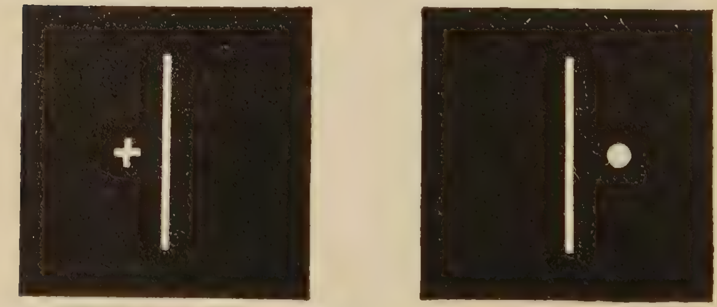

FIG. 82.—Simultaneous macular perception.

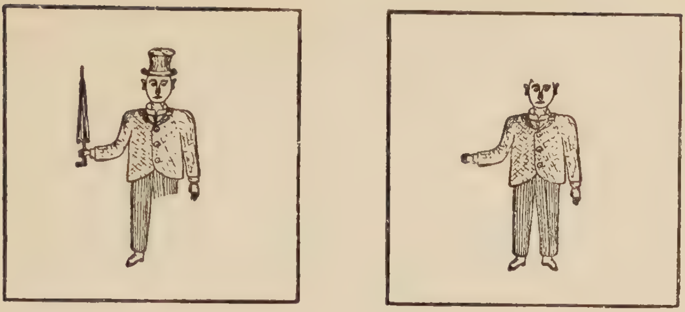

Fig. 83

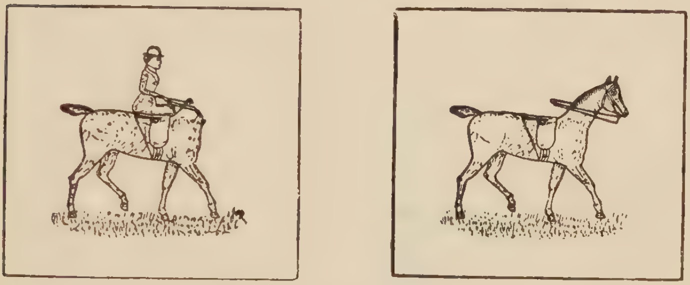

Fig. 84

Figs. 82, 83 and 84.—Fusion

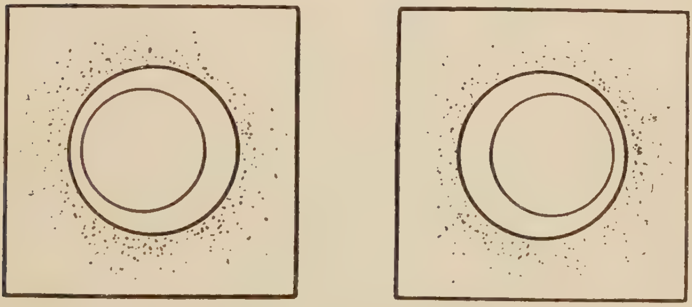

Fig. 85.—Depth perception.

Figs. 80, 81, 82, 83, 84, 85.—Fusion charts for use in the amblyoscope. (Courtesy of Claude Worth and Baillière, Tindall & Cox.)

can be accomplished, in all probability fusion is either absent or so defective that it cannot be trained. If it cannot be trained, only cosmetic results may be hoped for. If, however, the second degree is found, namely the presence of some degree of fusion, the hope of ultimate perfect results is increased.

The presence of the third degree of fusion, a sense of the third dimension can be determined by Mr. Berry's circles previously referred to, the principle of which is incorporated in Fig. 85. When fused properly, a bucket or tub or basket may be seen right side up, or upside down, as the case may be. This gives one the absolute assurance that, with proper management and proper coöperation of the parents and the child, if old enough to coöperate, ultimately single binocular vision may be obtained, in some instances without operation, but more often with operation.

To Claude Worth, for his insistance that this study with the amblyoscope is feasible in most cases and of infinite value in the proper management of squint, the profession owes a debt of gratitude. One looks back with regret over histories of cases of some years ago. Cures were for the most part cosmetic. Many of these cases, if managed in the light of our present knowledge, might have been included in the list of cures, *i. e.*, restoration of single binocular vision.

Treatment of Monocular Esotropia.—Inasmuch as all forms of concomitant squint have points in common as to symptoms, etiology, and treatment, the management of monocular esotropia will be discussed in detail. Before treatment is undertaken, a broad survey of each case is necessary. Such treatment as is indicated and possible should be instituted at the earliest possible moment. In the main, three objects are sought: (1) The correction of refractive errors if such errors are of sufficient degree to warrant correction. (2) The prevention of amblyopia exanopsia, or the correction of the same if it has developed. (3) Training of the fusion faculty if it is present. In order to carry a case through to a successful issue, with or without operation, these three objective points must receive due consideration. The student will naturally inquire if careful attention is paid to the three essentials just cited, is it possible to correct monocular esotropia without operation? Undoubtedly a percentage of cases do recover with restoration of parallelism of the visual axes and single binocular vision. The author is not prepared to state in definite terms what percentage of his cases have had these happy results without operation. The conditions which make this result possible are increasing, or rather the barriers which prevent restoration of single binocular vision and parallelism of the visual axes are becoming fewer or more easily controlled during the last decade of improved technic. Suffice it to say that one may hope by the adoption of systematic measures to effect a reasonable number of cures without operation and that many more real cures may be expected with operation than were observed ten years ago.

Refraction. Although the discriminating family doctor may refer his little squint patients to the specialist as soon as the squint is definitely established, the correction of refractive errors by glasses must be postponed until a suitable age. The youngest age at which the author has prescribed glasses successfully was sixteen months. An estimation of the refractive error should be made as early as possible. When the patient is under one year, a fair estimation of the error may be obtained by means of the ophthalmoscope. If the error is high, 3 diopters or more, this preliminary study will guide the surgeon to make a more accurate study by means of retinoscopy under a cycloplegic. If the error is high and particularly if anisometropia is present, suitable glasses should be prescribed from the retinoscopic findings just as soon as the surgeon can satisfy himself of the correctness of his estimation of the error and the child will tolerate glasses. In the author's experience, it is a mistake to postpone the prescribing of glasses until the third or fourth year, if the mother's coöperation can be secured. It is true, measures may be adopted at this early age to prevent deterioration of vision in the squinting eye, but a properly prescribed pair of glasses furnishes the greatest safeguard against this tendency, if the errors are high and unequal. Furthermore, if the esotropia is purely of the accommodative variety, good vision in both eyes is thus guaranteed. At the same time, elimination of the excessive convergence which is due to over-accommodation is secured by the wearing of glasses and the visual axes may be restored to parallelism. A third advantage to be obtained by the early use of glasses is gain in the development of the fusion faculty.

The best cycloplegic for all children under ten years of age is atropine sulphate. Children over five years of age will tolerate a 1 per cent solution while in younger children a 0.75 per cent solution may be used. The drops should be instilled three times daily for several days before an examination is made. If due care is observed in compressing the puncta after instillation of the drops no toxic symptoms will appear. Retinoscopic examinations should be made on successive days until satisfactory measurements have been made and a full correction is then prescribed. A moderate deduction of 0.25 or 0.50 diopter may be made in high errors. There is something to be gained by continuing the use of the atropine for several days after the glasses are adjusted. The better vision obtained by the glasses while the ciliary muscle is inoperative, convinces the child that he sees better with the glasses on, and in addition atropine holds accommodation as well as convergence in abeyance.

How often should the refraction be repeated? In young children every six months; in older children each year until one notes that the error is stationary. It is difficult to lay down principles in this matter which will apply to all cases. The above statement will apply to many cases, but in this matter as in refraction in general, the surgeon should be guided by the individual indications of each case.

Prevention and Treatment of Amblyopia.—It was pointed out that amblyopia is the greatest barrier to the successful treatment of squint. Furthermore, it is of vital importance to each patient as to whether he shall go through life with full vision of both eyes, or to all intents and purposes blind in one eye. The second step in the treatment of squint is one of much importance. If the child is too young to wear glasses, this step may be the first to be instituted. It is not contraindicated at any age. The best results are obtained in the early years of childhood. This treatment consists in forcing the child to use the squinting eye for definite periods each day. We have recourse to two methods, namely the use of atropine in the good eye or occlusion of the good eye for some hours each day. The latter method is the better.

Atropine Method.—In this method, atropine is instilled into the good eye once or twice each day for several weeks. It is expected that the child will elect to fix with the squinting eye if this is done. It is not always successful The author has observed children to continue to fix with the good eye even though atropine was used. The method is not without some risk in the average home. On the other hand, if the method is successful in transferring fixation from one eye to the other, care must be observed not to continue the treatment over too long a period lest the amblyopia be transferred from one eye to the other. If this method is employed, the child should be under the observation of the surgeon in order that a check-up may be made as to the state of vision.

Occlusion of the Fixing Eye.—In the author's experience, this is the most satisfactory plan to follow. The mother should be instructed to occlude the good eye by a bandage and cotton for definite periods each day, from two to six hours. The closing of the good eye must be complete. Very young, as well as older children, will endeavor to peep from under the bandage if it is possible. In cases in which amblyopia has not appeared, a two-hour session each day will be sufficient. If amblyopia is present, four to six hours daily may be required. In a few instance, if may be necessary to occlude the fixing eye continuously for several days in succession at the beginning of the treatment. When fixation has been partly lost, the child at first finds it difficult to avoid falls, and to properly orient itself. In the more difficult cases, it is best to begin play at close range with blocks and toys of fair size. If the child knows his letters, books or blocks of large type or drawing on the blackboard furnishes good exercise to command fixation and stimulate macular visual acuity. After a few days, the bandage is borne without much protest. In children old enough to go to school, the author has always insisted that the occlusion bandage be in place during part of the school period. At the first visit, the child is asked to write or print his name on a line drawn on a blank sheet without occluding the one eye, and then with the fixing eye occluded. It is a matter of great surprise as to the rapid progress which the child makes. Copying at home or in school insures rapid development and it is therefore made a routine part of work during the occlusion period, if conditions are favorable for its practice. Two hours spent at close work, in copying or in study with large type, will accomplish more than a four-hour period at play. Observations should be made at least once a month to check-up on the vision. If the acuity can be brought up to the normal or approximately normal, the period of occlusion can be made less each day.

The important fact to remember is this: *As long as monocular squint exists, the fight against amblyopia must be maintained in every case until the deviation has been corrected by operation.* It is not necessary for the surgeon to guide every step of this treatment. Parents can be instructed in the matter and their observations can be confirmed by the surgeon's examinations at stated intervals. In addition to this method of treating amblyopia exanopsia, the author, in association with Dr. Maxwell R. Bochner, Toronto, Can., added to the treatment the application of the "high frequency" current for eight-minute periods three times a week, with definite improvement in a majority of cases, and some improvement in practically all cases. In each instance, the usual method of treatment as outlined was carried out. Subsequently, the "high frequency" was added with marked improvement. The conclusions which seem to be justified are: (1) Amblyopia exanopsia is a functional condition, as the term implies; (2) under suitable treatment such amblyopia can be improved or corrected; (3) the improvement is greatest in childhood, but is observed in adolescence and early adult life.

Training of the Function Faculty.—The first and second steps in the management of squint can and should be practised at a very early age. Investigation and training of the fusion faculty can be begun between the ages of three and four years. If the refractive error has been properly corrected and measures are instituted to prevent or to correct amblyopia, the surgeon can await with safety the age when fusion studies can be made.

In a majority of instances a preliminary survey of the fusion faculty will definitely classify any individual case. As previously stated, unless some fusion tendency is present, in addition to simultaneous macular perception, time spent in training will be useless. If, however, even a moderate tendency can be elicited, patience will be rewarded by a reawakening and development of this faculty. For this purpose, the Worth-Black amblyoscope is used. The addition made by Black to the instrument consists in the rheostat attachment which enables the operator to dim the object before one eye and brighten the other. This is an ideal addition but not strictly essential. The same object may be accomplished by exposing one tube to a bright light while the other is more or less dimmed by the hand or a card. The test should begin with the child on the mother's or surgeon's lap, and the test should be converted into a play of catching a rat in a trap, a bird in a cage, etc., the tubes having been placed at the proper angle to make the two objects simultaneously perceptible. From these simple cards, one proceeds to other cards which require fusion. Contrary to what one would expect, the third dimension can be elicited in young children very readily. Mr. Berry's addition to the stereoscope can be adapted to use on the amblyoscope. It is not difficult for the child to convert the apparently meaningless circles (Fig. 85), into a real bucket or tub. By changing the cards about, the tub or bucket may be made to stand on its bottom, or reversed. By a little practice, the child will be able to tell definitely whether he sees the bottom or the top. Unfortunately the series of cards which are supplied is not sufficiently large to keep up the interest of the child. However, the mother is instructed in the use of the amblyoscope and the exercises are practised regularly at home. While it would be advantageous to have a larger series of cards which would call for the exercise of fusion, even the use of the cards of the first series (simultaneous macular perception) are of much value. Each of the cards requires precision and the exercise of binocular vision.

A ten-minute session each day serves as a sufficient stimulus to the fusion faculty to become active and to respond to the more valuable stereoscopic exercises after operation. The desire for fusion grows with surprisingly little stimulation at this early period, and with it, in some instances, the squint disappears. More frequently, the squint persists, but with the reëstablishment of an active fusion faculty, the prospects of a perfect recovery after operation are assured.

The cards as shown in Figs. 80 to 85 are arranged in groups, 1, 2 and 3, the order in which they should be used first to establish the state of the fusion faculty, and second, to develop it. Series 1 may be practised with great advantage at each sitting. However, if series 2 and 3 can be combined properly, a more substantial gain may be made in strengthening fusion.

One naturally asks if the measures as outlined are carefully practised, should we not expect a larger number of spontaneous cures without operative interference? In a majority of instances, one notes a dimunition in the angle of squint after glasses are worn, but the instances of complete disappearance of squint with glasses alone are comparatively rare. The accommodative factor in many cases may be an important one in the cause of the squint, but the angle of deviation is far in excess of what might be expected from the refractive error alone. The absence of fusion control is responsible for the break, but other factors determine the extent of the deviation —a deviation which in most instances is far in excess of the breadth of even a normal fusion faculty. As pointed out, the muscles, fascia, and check ligaments, when fixed by several years of definite deviation, offer an obstacle in molecular and anatomical changes which may require surgical intervention to be brought within the scope of fusion. It, therefore, is not unusual to find many cases which require more than a correction of refractive errors, and an awakening of the fusion faculty, to restore a normal muscle balance. The cures which are effected by these measures alone without operation occur before the sixth year. On the other hand, one cannot fail to be impressed with the necessity for very accurate correction by operation in older children and in adolescence. It is a matter of frequent observation that, even though all the measures suggested are practised with care, in mature children, unless the operation is carefully performed, perfect alignment cannot be obtained. The author has observed that some of these patients after operation can fuse any of the stereoscopic charts in the Wells' series, and yet in actual routine use, there remains a deviation of from 5 to 10 degrees. After further operative measures, so that almost perfect alignment is secured, the fusion faculty finally dominates binocular vision, and single binocular vision is secured. The reasons for this fact are to be found in the organic changes which have taken place in the muscles and fasciæ, and in the inelasticity of the fusion faculty. In these reasons, therefore, one finds the importance of early training as well as early operation, if this measure becomes necessary.

Operation.—If operation becomes necessary, the surgeon is confronted with two interesting problems: (1) At what age shall operative interference be practised? (2) What operation shall be performed?

There is a general tendency to postpone operation as long as possible, in fact too long to hope for the restoration of single binocular vision. The average age when squint operations are performed is from eight to twelve years. More are operated on after ten than before ten years of age. In the author's judgment, this is a mistake. Aside from the slightly increased difficulty in the surgical technic experienced in operating on young children, there is no contraindication to early operation, but the opposition of parents. Nothing can be gained by delay, and there is much lost. It is alleged that a child twelve years of age can be operated upon under local anesthesia. This is possible in many instances, but the gain which accrues to operating under local anesthesia is more than overbalanced by the fact that the operation is less well performed at this age than when the surgeon is freed from handicap by the use of a general anesthetic. As much as it is desirable, therefore, to operate under local, the average patient twelve years of age will do better under a general anesthetic.

Advantages to be gained by early operation are: (1) Macular vision is less likely to suffer; (2) the fusion faculty is more responsive to stereoscopic training after operation at the early age of six years. These two reasons are sufficient in themselves to commend early operation. There is another fact which should receive more consideration than is usually accorded to it. The disfiguring characteristic of squint, subjects these little sufferers to the ridicule of other children and to the common nickname of "cockie." The author has repeatedly heard from his little patients the following comment: "They can't call me 'cockie' any more, can they, doctor?" The mental distress occasioned by the deformity is not confined to older children and young adults, but it is repeatedly observed in quite young children.

Finally, if one hopes to restore the full and normal function of the eye muscles which is required in single binocular vision, it is best to begin early, when conditions are more favorable and before the overtaxed period of school life is begun.

Choice of Operation.– From a purely surgical standpoint, this is determined by the angle of deviation, the presence or absence of amblyopia, and the state of the fusion faculty. For reasons which are difficult to understand, tenotomy of one or both interni has held sway in the past and is even practised by many today. The operation has nothing to commend it unless it be a modicum of surgical skill required in its execution. To offset this advantage, if it can be considered as such, is first, the danger of converting a convergent esotropia into a divergent paralytic squint. If the patient escapes this disaster, he may be confronted with an equally serious consequence, namely, inability to sustain convergence at the near-point. In addition to these serious objections, there remains the fact that in those cases in which reattachment may be sufficiently secure to prevent an outward deviation, the most that may be expected is approximately a correction of 11 degrees. Many who continue to practise this operation, justify the procedure by pointing out the fact that they do not aim to cut all the spreading fibers of the muscle. The fact remains, however, that in order to obtain even 10 degrees of improvement in the squint, the tendon must be severed freely enough to allow the muscle to become retracted by its own elasticity. All who have observed the serious consequences of tenotomy of the internal rectus and have attempted to search for and reattach a muscle so tenotomized, will need no word of caution against a procedure which may truly be called bad surgery. Those whose experiences are somewhat limited, will do well to acquire the technic of the other measures to be described, rather than adopt a measure which requires no special surgical skill, but is fraught with so much danger.

The constructive surgical procedures to which the surgeon may resort are resection, with or without advancement, tucking and recession.

Resection.—It is not our purpose to describe the technic of these several operations, but certain aspects should be considered at this juncture, in order to intelligently determine the indications for each procedure.

A resection consists in excising a section of the tendon and muscle, and reattaching the distal end of the severed tendon to the tendon's stump. It is obvious that by this procedure no increased purchase is obtained upon the eyeball in external rotation. The tendon is simply shortened to the extent of the section excised. The contact arc of the muscle to the globe has not been increased. Under average conditions approximately 10 degrees of correction may be gained by this operation. In more favorable cases a slight increase over this amount is obtained, but too great a resection will place too much tension upon the muscle and a tendency to enophthalmos is encouraged. Furthermore, the tension placed upon the sutures is increased and the ultimate result may be less than that obtained at the time of operation. This operationis suitable, therefore, for squint of 10 to 12 degrees. When a bilateral resection is performed, one may hope to overcome about 20 degrees of convergence.

Advancement.—Much stress has been laid upon the contact arc of the muscle by many surgeons. In order to increase the contact arc, in addition to resection of the tendon, the distal cut end is attached not to the stump, but is brought forward and attached to the sclera close to the cornea. Before attaching the muscle, the stump is carefully trimmed in order that the muscle does not become attached at this point. It is held by some that attachment between the globe and the advanced muscle does take place at this spot, and that therefore there is no increase in the contact arc. Worth believes that such attachment does not take place if the operation is carefully performed. The author has no experimental data to add to Worth's testimony on this point, but his clinical experience confirms the evidence of Worth and others that a higher degree of correction may be obtained by a resection with advancement than with a resection alone. It is possible that this gain may be due solely to the fact that the muscle is shortened to the extent of the advancement in addition to the gain by resection. If this is true, clinical experience teaches that an advancement is more satisfactory than a resection. To obtain the same degree of correction by a resection, the entire tendon of the external rectus must be sacrificed and the muscle itself must be attached to the tendon stump. The results are less satisfactory than when the tendon itself can be reattached to the sclera in front of the stump. There is less occasion for the sutures to cut through and minimize the results.

If more than a gain of 10 degrees is desired, advancement should be added to the resection. One can hope to gain about 15 degrees of correction in an average case and more under most favorable conditions. By advancement of both external recti, 30 to 35 degrees of esotropia may be corrected.

Tendon Tucking.—Tendon tucking consists in folding the tendon by one of various methods, suturing the same and thereby shortening the tendon and muscle. The results, in shortening the muscle by this measure, are exactly the same as one observes in resections. Enthusiasts claim that almost any degree of shortening may be obtained by this method. As one writer expressed his views, "one can go back and take additional reefs in the muscle until the desired amount of shortening is obtained." The author cannot say that his experience with this operation has been so happy.

The two phases of the operation which commend themselves in the correction of moderate degrees of squint are: (1) The muscle is not severed; (2) the convalescent period is shorter than in resections and advancements. The long period of occlusion of both eyes required by an advancement is very much shortened in tucking. There are minor objections to the operation, but the chief objection is the same as that of resection. It is applicable only to squints of low degrees.

Tendon Recession.—Recession of the internal rectus, proposed by Jaimason,[1] consists in attaching the cut tendon 1 to 5 mm. back of the stump. It is an operation which should always take the place of a tenotomy, and may be used to advantage, combined with an advancement, in the higher degrees of squint when it is desirable to limit the surgery to one eye, or when double advancements are insufficient to correct the entire amount of deviation. On general principles it is inadvisable to weaken the converging power of the eyes when there is even a remote possibility of restoring single binocular vision. It is generally believed that it is better surgery to strengthen the externi either by resection or by advancement. The recession operation should, therefore, be held in reserve as a supplementary procedure to increase the effect of single or bilateral advancements.

Grouping of Surgical Cases.—With few exceptions, all cases in need of surgery may be classified in one of two groups. In the first, or cosmetic group, should be included all cases of high degrees of amblyopia and those in which the fusion faculty is totally absent. In this group, if the squint is not too great, not over 35 degrees, the surgery may be limited to the squinting eye. In the second group, or potentially successful group, should be included cases of normal visual acuity in both eyes, or correctable to normal by means of glasses, and cases of a normal or moderately defective fusion faculty. In either group a deviation of more than 35 degrees may call for special modification of the surgical measures advised.

Cosmetic Group.—If amblyopia is of considerable degree and hopeless, or if the fusion faculty fails to respond to training, it is best to limit the surgery to the squinting eye. By means of a free resection and advancement of the external rectus, one may hope to overcome a maximum degree of convergence. A resection alone will rarely yield more than 10 degrees. It is true that under certain conditions more than 20 degrees of deviation may be overcome, but in the judgment of the author, a greater gain than 20 degrees is not the rule. Too great a resection will tend to place too much tension on the muscle and will tend to create enophthalmos. If a greater correction is necessary, the surgeon may choose between recession of the internal rectus at the same sitting, or advancement of the other external rectus at a later date. The former method is preferred. It obviates the necessity of a second operation. The internal rectus may be set back 3 to 5 mm. with excellent cosmetic results. It is understood that in this type of case only cosmetic results may be obtained. Furthermore, if the fusion faculty is absent, or amblyopia is present, sustained convergence for close work need not be considered.

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[1] The author has practised recession of the internal rectus for a number of years using a method similar to that proposed more recently by Jaimason.

If the squint is slight, about 10 degrees, instead of a resection with reattachment of the muscle to the tendon's stump, a tucking of the external rectus may be practised. This operation is received with favor by some surgeons. No greater gain can be obtained by this operation than by a simple resection. The convalescent period is less prolonged than is that of a resection. Furthermore, the surgical skill required is less than is that of a resection. It also has its disadvantages which have been discussed. Instead of a tucking of the external rectus for moderate degrees of squint, a recession of the internal rectus may be practised as a third choice. The latter operation is not recommended in this instance.

Potentially Successful Group.—In the second group, the fusion faculty may be present, but defective or fairly well developed. Amblyopia is not in evidence. If the squint is of moderate degree, 10 degrees, a resection or tucking may be practised. Twenty degrees of deviation indicates a free resection and advancement. Deviations of 20 to 35 degrees should be corrected by double advancements. Some may elect to confine the surgery to the squinting eye and perform an advancement of the externus and a recession of the internus. In this group of cases, these procedures are not so satisfactory as double advancements. If the angle of squint is over 35 degrees, double advancements and recession, of one or both interni, is indicated.

When double advancements become necessary, the question naturally arises, shall they be performed at the same time or shall the second operation be performed after an interval. Circumstances, such as the convenience of the patient, etc., may decide this question; but when it is possible, it is good judgment to operate in two stages, allowing a period of about ten days to intervene.

Stereoscopic Training. Following operation in the second group, if proper alignment has been obtained, the amblyoscope should be replaced by the stereoscope. This becomes the last step in the management of cases included in this group. Many correctly performed operations fail to restore single binocular vision because the surgeon fails to follow his case after operation with this very essential step. The amblyoscope possesses great possibilities in stimulating a sluggish fusion faculty to activity, but it does it imperfectly. The stereoscope on the other hand is an instrument of great refinement, not only in training the fusion faculty to the point when it will function without further aid, but in stabilizing a muscle balance which was entirely absent throughout the squinting period. To briefly summarize, the role, which the stereoscope plays in this particular type of case, is that of deciding whether the result shall be only a cosmetic one, or a success in the truest sense of the word—restoration of single binocular vision.

ALTERNATING ESOTROPIA.

Etiology. – In the alternating type of convergent squint the patient, at will or unconsciously, fixes with either eye. The fusion faculty is absent, or so defective that training does not improve it. It begins at an earlier age than does the monocular type. Squint is evident in some cases at birth, or in the first few weeks of life. A majority of cases are well established before the end of the first year. As the patient uses both eyes alternately, amblyopia does not develop.

Refractive errors may or may not be predisposing factors. In a majority of instances, the refractive error is so moderate that it can in no way be considered as a cause. In other instances, the error may be considerable, from 2 to 4 diopters. Unequal errors are rarely observed. On the whole, refractive errors play an indifferent role.

The influence of heredity probably consists in the transmission of a defective fusion faculty. Other precipitating factors, so frequently observed in monocular squint such as fright, convulsions, and acute illness, are observed, but not so frequently as in the monocular variety. More often, the onset is insidious without any such associated conditions.

The chief etiological factors are two, an absent or undeveloped fusion faculty and heredity. As the hereditary influence is shown mainly in the inheritance of a weak or an absent fusion faculty, one might say the causes of alternating esotropia are practically reduced to a single factor.

Course of Untreated Alternating Esotropia.—The absence of fusion, of amblyopia and of marked refractive errors, simplifies the course of alternating squint. With probably one exception, nothing changes the character of an untreated case throughout life. The patient goes on squinting through life with little alteration. The one exception to this general rule is the conversion of an alternating squint into a monocular one. If the refractive error is appreciable, if there is even a slight difference in the visual acuity with or without an appreciable refractive error, the patient may give preference to one eye and use it to the exclusion of the other. A monocular type of squint may therefore develop with all the characteristics of monocular squint.

Myopia instead of hypermetropia is occasionally observed. This is noted in older children. It occurs, however, in probably the same relative frequency as the condition is observed in non-squinters. It is to be remembered that hypermetropia and astigmatism are often very moderate in degree. The same process, the passing from hyperopia into myopia through the "turnstile of astigmatism," referred to by the late Dr. Samuel Risley, may be observed in alternating squinters as well as in non-squinters.

Diagnosis and Measurement. In the history of the case, one usually receives the definite information that the squint appears in "both eyes," by which the parents mean, the squint shows now in one eye and again in the other. A careful cover test will definitely reveal the alternating character of the esotropia. This and the measurement of the angle of the deviation were thoroughly discussed in monocular esotropia and need no further amplification at this time.

Treatment.—Insofar as refraction is concerned, the same careful methods are practised as were described under monocular esotropia. The greatest care should be observed that glasses are prescribed early, if the error is regarded as sufficient to be an etiological factor. A spheric error of less than +2 diopters would hardly be regarded as high enough to require correction before the child begins its school work. Astigmatism of +1 diopter or more should be considered more seriously, and a correction should be worn as early as possible. All higher errors, spherical and cylindrical, should be fully corrected. In discussing the etiology, the minor role of refractive errors was referred to. They should not be ignored, but should be given the same consideration as is given them in non-squinters. This applies to moderate errors. Higher degrees should be treated as we have previously outlined.

Amblyopia.—Amblyopia is not observed in true alternating squint. No special measures need be practised, therefore, to prevent its development, other than a careful check-up, at stated intervals, on the visual acuity in both eyes.

Fusion Training.—Fusion training, like amblyopia, is unimportant in this type of squint. One approaches this step in the management of alternating esotropia as in the matter of refraction. As soon as the child is old enough, an effort should be made to classify the case as to the presence or absence of fusion, and if fusion is present, to determine its degree. If one can demonstrate to his satisfaction that the case has more than "simultaneous macular perception," every effort should be made to develop even a very defective fusion faculty. To this end the amblyoscope should be used with as much care as it is employed in monocular esotropia. Inasmuch as many of these patients squint soon after birth, it is probable that any fusion which may have been present fails to develop because of the barrier created by the squint. To all intents and purposes, the ultimate results may be the same as when fusion is totally absent. When, however, squint appears later in childhood, a fusion faculty although undeveloped, may persist and yield to training. In the author's experience, few cases respond. The effort, nevertheless, should be made in every case in which one finds even a suggestion of fusion present. When totally absent, or at least unresponsive, as it is found in a majority of instances, fusion training can be omitted.

Operative Treatment.—Every case of true alternating squint becomes a potential surgical case from its onset. Little hope of correcting the squint without operation can be held out to the parents. The question becomes one of "when shall the operation be performed?" The operation is solely a cosmetic one. Contrary to the usual custom, the author believes that it is to the best interests of the patient to operate early, between the fourth and eighth year. So long as the deformity is to be corrected, early operation relieves the child of the annoyance which the disfigurement creates. Furthermore, the temporary diplopia which is sometimes experienced by older children and young adults after operation, does not seem to inconvenience the young child. A third advantage in early operation is found in those cases which require several operations, in that the first operation improves the general appearance of the child and the second operation may be postponed for a year or two. The most serious objection to early operation is the greater difficulty in the general management of the young child as compared with one who is more mature. In the average case this is more than balanced by the advantages gained by correcting the defect before the child starts on its educational career.

Choice of Operation.—This may be determined entirely by the degree of squint to be corrected. All operations in this group are cosmetic and one hopes simply to obtain perfect alignment. The question of near-point convergence becomes secondary. The surgery may, therefore, be confined to one eye or bilateral advancements may be performed. In the matter of the degrees of correction to be expected from each procedure, the same general principles apply as in that of monocular esotropia. In a general way, 10 degrees of deviation call for a moderate advancement, a simple resection or a tucking of one external rectus. Twenty to 25 degrees require an advancement with a recession of the corresponding internal rectus, or a double advancement of the externi. Any deviation beyond 25 degrees will require a double advancement with a single or bilateral recession of the interni. In the high degrees of deviation, the best method of procedure is to obtain maximum results by performing an advancement and a recession on one eye at the same sitting. Any residual squint is corrected at a later period by a similar operation or operations on the fellow eye. As explained previously, it is inadvisable to operate on both eyes at the same time.

It is quite as important to make perfect corrections in this form of squint as in monocular esotropia. The author has always found it advantageous to proceed slowly. This method insures greater precision and exactness in results. If the first operation does not fully correct the squint, a second one can later be performed on the fellow eye.

Over-correction.—The question is often raised as to whether the operator shall aim at over-correction. This may be answered in the affirmative, and in the negative. Affirmatively, because one rarely obtains the full correction which he hopes to reach by any single procedure. In this sense, therefore, one should always aim at over-correction, and the results usually will be satisfactory. It may be answered negatively in another sense. Over-correction is often advised because the stitches cut through and an advancement, for example, may not remain where anchored to the sclera. If the very satisfactory Worth advancement is selected and the stitches are properly anchored into the episcleral tissues, one usually finds the ultimate result continues to be the same as when the operation is completed on the table. Slipping of the sutures and recession of the advanced muscle is usually due first to improper placing of sutures and second to the attempting of too great a correction by a single advancement. Attention was called to the danger of attempting to correct too much by an over liberal resection and advancement in the hope of obtaining full correction by a single operation. The result usually is an under-correction, because of the great tension placed on the sutures which thereby allow the tendon to recede. If the operation is properly performed, and the resection is correctly calculated, no attempt at over-correction in the sense referred to, need be considered.

Postoperative Diplopia. -The question of double vision after operation has not received much discussion. It rarely causes inconvenience during childhood. The habit of suppressing the vision of one eye becomes habitual and the average young individual rapidly readjusts himself to the changed relation of the two eyes, and diplopia as a rule is hypothetical rather than real. After operations, during adolescence and early adult life, one occasionally meets with diplopia. The author recalls but one instance in a young woman, aged twenty-three years, in whom diplopia was an annoying symptom for some weeks after operation. Conscious diplopia, as a rule, rapidly disappears and fear of developing the same after operation should not deter the operator from undertaking the operation upon adults.

EXOTROPIA OR DIVERGENT SQUINT.

Divergent strabismus differs in etiology, in its course of development and in treatment from that of esotropia. It occurs in a majority of instances about the age of puberty or during the adolescent period, although the "essential" variety may be observed in young children. Unlike esotropia, which frequently diminishes by the constant wearing of glasses, exotropia tends rather to increase than to diminish as time goes on. Furthermore, the angle of deviation is less constant in degree. It does not respond to non-surgical methods of treatment so readily as does esotropia, and surgical measures are less satisfactory than in the convergent type of squint. To offset these unfavorable aspects of divergent concomitant squint, there remains the fact that exotropia is of less frequent occurrence than is that of esotropia.

Classification. The grouping of cases of exotropia in the main, is similar to that of convergent squint, but a trifle more complicated. It may be monolateral or alternating in type. Amblyopia is comparatively rare. The alternating type is probably more frequently observed than the monolateral type. If those cases, which are due to very defective vision in one eye, are excluded, the alternating type predominates.

In a way, one may further classify exotropia as *accommodative,* or to speak more to the point *myopic,* and second *essential.* Worth applies the term "neuropathic" to the latter group, but admits that he uses the term for want of a better one. In this treatise, "essential" will be used to harmonize with the classification in esotropia. There are types of exotropia which do not clearly fall within either of the two groups mentioned. The divergence which so often follows tenotomy of the internal rectus is not strictly speaking a paralytic squint, as we know paralytic squint. To give it the prominence which it demands, it will be called *postoperative exotropia.*

Myopic or Accommodative Exotropia.—Strictly speaking, it is the absence of accommodation which is a pronounced factor in myopic exotropia. In this type the fusion faculty may be present and well developed. Myopia is gradual in onset and often reaches 5 or 6 diopters before a tendency to divergence appears. No precipitating factors are noted, but exophoria especially at the near-point develops and as the near-point approaches, efforts to see with both eyes give way to monocular vision. Divergent squint becomes an established fact. It may be unilateral or alternating. It rarely becomes strictly monocular unless marked anisometropia develops. This type often is only moderate in degree, from 15 to 20 degrees. At times it may almost disappear in distant vision. The cover test, however, will definitely show the covered eye deviates outward. In other instances, if myopia is high, the deviation may become considerable. This is similar to the conditions observed in malignant myopia, when the elongated eyeball together with the developing orbit, brings about an exotropia of varying but usually of considerable amount. In this group may be included many cases of exotropia due to gross visual loss or blindness in one eye. Most blind eyes tend to outward deviation when blindness or visual loss develops after the age of puberty.

Essential Exotropia.—The student will recall, in the section on Exophoria, that although it develops, as a rule, after the age of thirty years, it occasionally is present quite early in life, and that it occurs in hyperopia as well as in myopia. Unless the condition is corrected, the exophoria gradually increases, and after a time, all effort to hold the divergence in check is abandoned and exotropia becomes established. If the refractive error remains hyperopic, as often is the case, the resulting exotropia cannot be said to be accommodative in type. It is an essential variety. Just what the etiological factors are, is difficult to determine. The fusion faculty is weak or absent in most cases, but present in others. In a larger number of cases which belong to this group, divergence is present in early childhood. The fusion faculty is defective. Refractive errors are low and may either be myopic or hyperopic. It is probable that a defective fusion faculty is the active etiological factor. The exotropia usually is alternating in type, although in some instances, the squint is unilateral. As is characteristic of exotropia, the angle of squint varies from time to time. Many of these patients can, without any effort, render the visual axes parallel, but when not consciously observant of the fact, a manifest deviation is present. One of the author's patients, a boy, aged eight years, can use his eyes perfectly together when he appears in the office under observation. He can fuse objects in the stereoscope without difficulty, but habitually allows the left eye to wander out about 15 degrees in all of his unconscious visual acts. He belongs to a type which Worth describes as, "bright, quick-witted, and intelligent," but "nervous and highly strung."

Postoperative Exotropia.—This is a name given to an unfortunate exotropia which so often follows a tenotomy of an internal rectus. In a minority of instances, the outward deviation follows soon after the tenotomy has been preformed. Such cases usually are of a paralytic variety and internal rotation is much restricted. The angle of deviation varies with the rotation of the good eye. In a majority of instances, however, divergent squint develops very gradually. and definitely appears several years after the tenotomy is performed. Most operators who were taught to perform tenotomies find a number of these patients return to them several years after operation with a divergent instead of a convergent squint. When the squint reaches a maximum degree, it too may resemble a paralytic squint with little power of internal rotation. In addition to the disfigurement, which is a source of much embarrassment, many of these patients complain of a constant eyeache or soreness. In all these cases, the tendon has not become reattached to the sclera but retracts within its sheath. The firmness of the capsule may hold the eye in position for a time, but a stretching process allows it to gradually turn outward more and more, and eventually the squint becomes a paralytic one. Inability of the patient to properly converge to the near-point, after the healing process of a tenotomy is complete, should lead the surgeon to anticipate the development of a divergent squint. Furthermore, one such case on his records should teach the lesson that tenotomies are dangerous and should be replaced by a recession operation, that is, an operation which sets back a tenotomized muscle by stitching it to a definite point on the sclera.

Symptoms.—Aside from the outward deviation of the visual axis, the symptoms and course of exotropia differ materially from esotropia. Esotropia yields to treatment in many instances by means of glasses alone and by the use of the stereoscope. Exotropia on the other hand, rarely tends to diminish, but usually increases. Neither glasses nor stereoscopic training tend to reduce the angle of squint. Amblyopia exanopsia is rare, but this fact does not render correction of the squint easier. The angle of squint varies. In fact, this variation adds to the difficulties which the surgeon encounters in the surgical straightening of the eyes. Diplopia is rarely observed. The fusion faculty is not defective in so large a percentage of cases as in esotropia, but its training does not add much to the treatment of the case excepting in the accommodative type.

Treatment.—All phases of the treatment of exotropia are less satisfactory than the management of esotropia.

Refraction.—The same care in estimating and correcting the refractive error, as was advised in esotropia, should be observed in any given case of exotropia. In the accommodative variety with myopia, a full correction, if supplemented by fusion training, may correct an exotropia of moderate degree. Unfortunately, as in esotropia, the amount of deviation is more than can be accounted for by the relation of the meter angle of convergence to the diopter of accommodation. Glasses, therefore, in themselves, rarely give sufficient relief to correct the squint. If the refractive error is hyperopic, instead of a full correction as is indicated in esotropia great care should be observed that the spheric error is under-corrected. In this, one must be guided by the patient's age and occupation. In young people, the sphere may be cut a great deal. After the age of thirty years, one should be guided entirely by the requirements of the patient's occupation. If the occupation is of such a character as to require the constant use of the eyes at the near-point, the age of the patient also becomes a determining factor. After thirty-five years, a full hyperopic glass may become necessary. Under certain conditions, therefore, the simple rules of refraction should be followed regardless of the presence of exotropia.

Fusion Training.—Classification of each case as to the state of the fusion faculty should be practised at the beginning of the treatment. Because of the average age of the patient, this is done with greater facility than is possible in the young esotropic patient. If the patient belongs to the group which shows a tendency to exophoria in the adolescent period, every effort should be made to overcome the exophoria by prism training as advised in the treatment of exophoria. If squint is definitely present, fusion training by means of the amblyoscope should be followed. The results are not encouraging but every effort should be made to restore a defective fusion faculty.

Occlusion of the Fixing Eye.—Many cases of exotropia are alternating in type and therefore vision is not apt to deteriorate. In the monocular variety, amblyopia exanopsia is infrequent. Occlusion of the fixing eye should, however, be practised as in monocular cases, unless the extropia is due to blindness or amblyopia other than that acquired from disuse. When one eye has a low refractive error and the other a high myopia error, with an outward deviation, nothing can really be done to correct the condition by any of the measures discussed.

Exotropia Combined with Vertical Disturbance.—The vertical balance in exotropia, as well as in esotropia, should be carefully determined. A hyperphoria, if present, should be corrected by prisms properly placed. A full prismatic correction should be incorporated in the glasses. One should determine the amount of hyperphoria for far and near, and the minimum full correction should be prescribed. The correction of the vertical balance often has a considerable influence over lateral disturbance, whether there is only a tendency to lateral disturbance or a manifest squint.

Operation.—Comparatively few cases of exotropia are corrected by non-operative measures alone. Before one proceeds to operative interference, each case should be carefully studied as to its particular needs. There are many cases of the alternating type, moderate in degree, in which the squint is neither disfiguring nor inconvenient to the patient. Some of these patients can use either eye at will, are not annoyed by diplopia and never complain of discomfort. Inasmuch as moderate degrees of exotropia are less noticeable than esotropia of the same degree, it is often inadvisable to recommend operation. The fusion faculty is defective and single binocular vision is not apt to follow after operation. Operative interference should be reserved for cases in which the deviation is sufficient to attract undue attention because of disfigurement, and for cases in which there is a reasonable hope of restoring single binocular vision. The latter result is less frequently obtained in divergent squint than it is in esotropia. The cosmetic operation, however, should unhesitatingly be resorted to if the deviation is definitely noticeable.

Fundamentally, the same principles apply in the surgery of exotropia as were discussed in detail in esotropia. There are some differences, however, which should be pointed out. A tenotomy of the external rectus, while not so apt to lead to subsequent paresis of the muscle, with secondary internal deviation, is of almost no value in correcting the outward deviation. Postoperative esotropia does occasionally follow complete tenotomy of the external rectus. It occurs less frequently than postoperative exotropia after tenotomy of the internus, but occurs with sufficient frequency to warn the surgeon against the practice of tenotomizing the externus. The author has operated upon several cases which came under his care for the relief of this surgical error. Advancement of the internal rectus, on the other hand, does not admit of as much gain as advancement of the external rectus. A number of reasons account for this fact. In the first place, the internal rectus is attached nearer to the cornea and one is therefore more limited in the shortening of the muscle by an advancement. In the second place, the operative field is more restricted and the surgeon encounters more difficulties. Third, the tendon of the internal rectus is short and the resection will necessarily include a considerable part of the muscle. Slipping of the sutures is more apt to follow. The young surgeon will, therefore, find that he cannot gain as much by advancing an internal rectus as he will in advancing the externus.

Tucking of the Internal Rectus.—Tucking of the internal rectus for the reasons just given is not advised. It is limited in its application to moderate degrees of the convergence. and in the author's experience, is of little value in divergent squint.

Advancement of one or both internal recti is the operation of choice. This may be combined with moderate resection of the same muscles, and with *recession* of the externi. In unilateral types, an *advancement* of the internus and a *recession* of the externus of the deviating eye should be practised. If these combined operations are not sufficient to fully correct the squint, an advancement, and if necessary a recession, of the same muscles of the other eye, should be practised after the lapse of ten days to two weeks. Both eyes should be occluded for at least five days after the operation in every instance when an advancement is performed. Following the operation, stereoscopic exercises should be prescribed if the fusion faculty admits of training and vision is good in both eyes.

Correction of Postoperative Exotropia. The surgeon will be called upon to treat many cases of this unfortunate and avoidable condition, so long as tenotomy of the internal rectus is practised. It would seem, from the number of these cases which one meets with in an average experience, that a correction of this condition should no longer be necessary. Protest from surgeons of repute have not been wanting in the past, but many allow these protests to go unheeded and continue to tenotomize. Some tacitly are conscious of the unsurgical character of the operation, and state that they do not aim to cut all the fibers, but allow the outermost strands to remain. Any surgeon of experience will testify to the fact that one does not see an appreciable gain after a tenotomy until all strands are cut. *If the tenotomy is performed thoroughly enough to bring about a dimunition in the squint, every restraining band will have been severed and the muscle will retract.* The student will, therefore, do well to discard tenotomies unless the cut end is stitched to the sclera at a point where it should become reattached. In a majority of instances, the outward deviation does not occur immediately, but comes on gradually *after the lapse of one or more years.* It is progressive and a deviation of from 50 to 60 degrees is not unusual.

The condition can be corrected even after the lapse of many years. General anesthesia is better than local. The conjunctiva over the internus is incised, and a careful search is made for the retracted end. This will be found imbedded in the capsule. A careful examination with the slit lamp has helped the author before operation to note approximately the location of the retracted muscle. The muscle should be grasped by forceps, detached, and brought forward with the capsule and should be reattached to the sclera. For this purpose, silk sutures are best. They may be brought out through the conjunctiva and tied. It is easier to operate on recent cases than those in which years have elapsed since the first operation. One can usually find the retracted muscle, however, even though years have elapsed. No other operations, as a rule, are necessary. If the squint is definitely a paralytic one and of long standing, it is advantageous to tenotomize the opposing and probably the organically contracted externus and by a recesssion, attach it further back near the equator.

In every instance, an effort should be made to find the cut muscle and reattach it. The results usually are good. If one fails to find the retracted end, the entire capsule in which the retracted end rests should be brought forward and attached to the sclera near the old tendon stump. The dissection necessary to free the conjunctiva from the sclera and capsule, helps to create a suitable raw surface to which the advanced capsule and muscle readily become attached. If this method of procedure is required, the opposing external rectus should be set back.

Diplopia Following Operation.—In the author's experience this symptom need not be feared. As in esotropia, if present at all, it is of a fleeting character. It should not be regarded as a contra-indication to operation, if the general character of the case indicates corrective surgical measures.

HYPERTROPIA.

A vertical concomitant deviation which exceeds the limits of hyperphoria is comparatively rare. In most instances, such a deviation is not strictly concomitant, but paralytic in character, and the surgery of vertical deviations will be discussed more fully in the section on Paralytic Squint.

Etiology.—The cause of most vertical deviations, non-paralytic in character, will be found in muscular anomalies, in faulty attachments, and in improper developments. A vertical balance is the rule at birth. Lateral deviations are normal in the first three months of infancy, or before the fusion faculty has become sufficiently active to maintain a lateral balance. The vertical balance, on the other hand, is present at birth. Furthermore, in an average case, little can be done by fusion to overcome more than 3 diopters of vertical deviation.

There is a type of hypertropia which is found in association with a similar type of eso- or exotropia in which the vertical deviation is as unstable as the lateral balance, a purely neurotic type of squint. It occurs in patients who have other stigmata of the neurotic element. The nervous system is totally lacking in poise and the conjugate innervations share in this instability.

Classification.—From an etiological standpoint, concomitant hypertropia may be divided into two groups: (1) Those due to muscular anomalies and (2) essential, neurotic hypertropia. Defective fusion does not play a prominent role in either type.

Differentiation.—One should carefully discriminate between the several types in this group and especially between concomitant and paralytic squint. Strictly speaking, the neurotic type is non-concomitant. The degree of deviation varies. But inasmuch as it is totally different in its etiology from paralytic squint, it more nearly conforms to the concomitant variety.

Treatment. Inasmuch as vertical deviations in this group of cases are usually found associated with lateral disturbance, the treatment is simply supplemental to the general management of lateral squint.

Refraction. Refraction is the same as in lateral squint with which it is associated.

Fusion Training.—Fusion training is of no avail. Little can be hoped for even in hyperphoria if more than 3 diopters and practically nothing in manifest vertical squint.

Prismatic Correction.—As indicated in the section on Hyperphoria, a vertical imbalance should be fully corrected by prisms. It is possible to incorporate 5 diopters of prismatic correction in each lens, making it possible, therefore, to overcome from 10 to 12 diopters of vertical deviation. It is inadvisable, to resort to surgery unless the vertical squint exceeds this amount.

Surgery.—If it is possible to determine whether the squint is due to an over-action or an under-action of one or more muscles, the character of the surgical indications is simplified. An under-action should be treated by an advancement. This applies to the superior and inferior recti. An over-acting muscle due to an improper attachment should be corrected by a recession operation. *A vertically acting rectus should never be tenotomized.*

Any residual squint, if considerable, can be corrected by operation on the opposing muscle of the same eye. For example, if advancement of the right superior rectus fails to correct a downward deviation of the right eye, a recession of the right inferior rectus may bring about the desired results. Any residual deviation under 10 diopters can be corrected by prisms.

Surgery is rather unsatisfactory, as are other measures, in correcting the neurotic variety. The fundamental condition is a lack of control. Basically, therefore, the treatment should be: (1) Constitutional, an effort being made to restore nerve balance; (2) amblyoscopic training; and (3) surgical.

In the first, indications are to place the individual in the proper environment, and to add to this such measures as may tend to restore nerve balance, if this is possible.

The fusion tubes are so arranged as to allow one to elevate one tube to a higher level than the other within limited scope. Practice may aid in limiting the ocular deviation. It rarely happens that the stereoscope can be employed, because of its limitations. However, if the squint can be brought to stable limits by suitable general and fusional training, operative measures may bring the deviation within reach of the stereoscope. As an end measure, if this is possible, the stereoscope may become of value. It will not correct vertical deviations, but tends to steady and restore balance. Fortunately, this particular form of hypertropia is rare.

Surgery, if practised at all, should be resorted to only after careful study and should be directed to the lateral deviations first, with the hope that with the restoration of lateral balance, vertical squint may be brought within the correcting power of prisms. If this is not possible, surgery of the vertical muscles may be attempted.

PART IV.

PARALYTIC SQUINT.

Paralytic squint, as it usually is called, differs so materially from concomitant squint that the two forms have little in common, aside from the dominating characteristic, namely, a deviation of the axis of one eye from its normal position in relation to the other. Even this deviation follows laws which are totally different in the two varieties. Notwithstanding the great dissimilarity of the two conditions, they are often confounded.

Etiology.—As the name implies, paralytic squint, essentially, is a form of strabismus due to paralysis of one or more of the extraocular muscles. The paralysis may be the result of disease or injury of the muscle itself, or of disturbance of the nerve which supplies the muscle. Instead of disease or injury of the muscles and nerves, an absence or an anatomical defect of these same structures may be the etiological factor. The latter condition is probably more common than is usually recognized, in spite of the fact that such conditions date from birth. Failure to recognize a congenital paralytic squint is due to several causes. In the first place, the history as given by the average parent is not always trustworthy from a scientific standpoint, and in other instances, because of the irregular behavior of the eyes shortly after birth under varying possibilities, the symptoms do not always admit of definite classification without a careful study.

Congenital Factors.—According to Whitnall[1] congenital factors are largely due to "errors in development by cleavage from the common premuscular mesoblastic mass." The anomalies found in the dissecting room and on the operating table would lead one to think that paralytic squint from this source is of more frequent occurrence than is recorded in the literature. There are, however, certain hereditary forms of paralysis which may not be due to errors of development. Congenital ptosis with paralysis of the superior rectus may be distinctly a familial type. Mr. E. Treacher Collins[1] regards some of these cases as an abiotrophy of the eye muscles. In some instances the degeneration is found primarily in the muscle fibers themselves. There may be no other symptoms than ptosis and paralysis of the superior rectus. In other instances bilateral ptosis may be associated with other symptoms. Of interest in this connection may be mentioned two unpublished cases of the author, congenital bilateral ptosis occurring in sisters, both of whom during adolescence developed a condition of myasthenia gravis.

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[1] Anatomy of the Human Orbit, p. 283.

It is interesting to note that congenital complete external ophthalmoplegia may occur without involvement of the ciliary muscle and the sphincter of the iris. An instance of this rare condition was reported by E. H. Cary.[2] Cruzon and Hague[3] reported a family of external ophthalmoplegia in three generations. T. M. Li[4] observed the same condition in three members of a Chinese family. Cary quotes Florenza's classification of total ophthalmoplegia, as follows:

1. Nuclear aplasia: Failure of the nuclei to develop.
2. Intra-uterine: Nuclear dysplasia—an arrest of development in fetal life, probably from an infection.
3. Natal dysplasia: Injury of the nuclei at birth.
4. Postnatal dysplasia from unknown causes up to five years, because the nervous system is not completely developed before that age.

That it is possible to find congenital external ophthalmoplegia with intact ciliary and sphincter muscle activity is explained by Cary by the fact that the nuclei of the oculo-motor nerve develop from different portions of the neural tube. The somatic nuclei may show arrest of development while the visceral portion may be intact and the sphincter of the iris and the ciliary muscle, therefore, are active.

Birth Injury.—Birth injury undoubtedly is responsible for many cases of supposed congenital ocular muscle palsy and paresis. There are differences of opinion as to how such palsies are produced. The greater frequency of paresis of the left external rectus, as recorded by Harold Gifford[5] would lead to the belief that even moderate pressure in the birth canal will explain many cases. In all probability many cases of hereditary external rectus paralysis can be accounted for by this means. It is interesting to record, however, that the last case reported by Gifford in his series was delivered by Cæsarean section, and that no evidence of injury was observed. When the paralysis is more wide-spread and not limited to a single muscle, nor even to one eye, it is probable that intracranial, and not peripheral trauma, is responsible. It should not be forgotten that increased intracranial pressure may injure the sixth nerve. Such increased pressure may be caused by direct compression of the skull, or by hemorrhage from within. When the paralysis is bilateral and other muscles are involved, some form of nuclear disturbance should be sought, either prenatal and inflammatory, or that brought about at birth by trauma.

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[1] Transactions of an International Congress, 1922.

[2] Contributions to Ophthalmic Science, dedicated to Dr. Edward Jackson, p. 122.

[3] Bull. et mém. Soc. méd. d. hôp., 1920, p. 372.

[4] Am. Jour. Ophthal., 1923, **6**, 816.

[5] Ibid., January, 1926, p. 3.

Acquired Palsies.—Birth injuries or obstetrical palsies really fall into this group, but inasmuch as they are acquired at the time of birth, we have included them in the congenital palsies.

In the group of factors which may produce ocular palsy by involvement of the muscle itself should be mentioned trauma. In the previous section, reference was made to "postoperative palsies" due to free tenotomy of the internus. Although a tenotomized internus is more frequently followed by paralytic squint than tenotomy of any other ocular muscle, the same condition has been observed after tenotomy of other muscles of the ocular group.

Perforating wounds of the orbit may sever one or more ocular muscles. Such conditions are not rare. Operations on the nasal accessory sinuses are occasionally followed by injury to one of the eye muscles. In radical operations on the frontal sinus, if the trochlea is not properly replaced, paralysis of the oblique is observed often enough to make the operator careful in the management of this part of the procedure. The author has studied at least one case of exenteration of the ethmoids in which an internal rectus was completely severed by punch forceps as a grand finale to the operation. Similarly, he observed a paralysis of the inferior oblique after excision of the lacrimal sac. deSchweinitz[1] reported a paralysis of the right internal rectus following one hour after an intranasal operation. At the same meeting, Greenwood placed on record an ocular palsy which followed a simple tamponing of the nose. There are instances of paralysis indirectly from trauma. Bordley[2] reported a Weber syndrome (abducens palsy, with crossed hemiplegia) which followed the improper introduction of a Eustachian catheter, a remote consequence of local trauma and probably infection. The lesion in this instance was intracranial, a pontile involvement in which the injury was to the nerve rather than to the muscle.

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[1] Trans. Am. Ophthal. Soc., 1926.

[2] Ibid.

Aside from surgical and accidental trauma, disease of the sinuses is responsible for a considerable number of ocular palsies, due directly to involvement of the muscles, or to involvement of the nerves. Although the disturbed muscle balance in most of these cases takes the form of a phoria, complete paralysis may follow in some instances. The reason for this possibility is to be found in the close relation of the sinuses to the orbital contents. The annulus of Zinn, which surrounds the optic foramen and forms the common source of origin of five of the ocular muscles, is in close juxta position to the sphenoid. The internal recti on the other hand, are in intimate relation with the ethmoids. The superior oblique has been observed to suffer in advanced frontal sinus disease; the inferior oblique bears an intimate relation to the lacrimal sac and groove the latter often undergoing necrosis during chronic dacryocystitis. Injury to and disease of the muscle itself occur less frequently than direct damage to the nerve supply. They nevertheless occur with sufficient frequency to be regarded as possible factors to be considered in searching for the cause of an ocular palsy.

A direct blow upon the eye is capable of producing a complete or partial external and internal ophthalmoplegia. This may be brought about by hemorrhage, or by direct injury to the nerves as they emerge from the skull into the orbit. It is a question, of course, as to whether the injury has a direct action upon the muscle structures or upon the nerves which supply them. Although the accompanying internal ophthalmoplegia (paralysis of the sphincter of the iris and of the ciliary muscle) might lead one to think that the injury may be direct nerve injury, the possibility of damage to the muscles cannot be definitely excluded. The fact that most of these cases eventually recover with complete restoration of function would tend to indicate that possibly direct nerve injury is the greatest factor. Very slight trauma to the cornea has been followed by loss of action in the iris sphincter at one point with subsequent recovery. This fact would lead one to the conclusion that the same may apply to the extra-ocular muscles. It is probable, however, that both muscles and nerves share in this type of traumatic paralysis.

Etiological Factors Affecting the Nerves. It is difficult to classify these causes. In the first place, one can group such factors with reference to the part of the neuron which is involved. To include the entire neuron such lesions may involve: (1) The higher centers in the brain cortex; (2) the communicating fibers which connect these higher centers with the basal nuclei in the floor of the fourth ventricle (injury or disease of these parts are manifest in the form of conjugate palsies); (3) the nuclear centers in the floor of the fourth ventricle; (4) the fascicular fibers which unite to form the several nerve trunks; (5) the nerve trunks; (6) branches of the nerves; (7) the peripheral parts of the nerves. In the particular phase of the subject under discussion, conjugate palsies will be excluded for the time, but will be discussed in a separate section. Furthermore, it seems best to arrange these factors from a clinical rather than from an anatomical standpoint.

Such factors may be: (1) Traumatic; (2) inflammatory and vascular; (3) neoplastic, and (4) toxemic.

Trauma.—Trauma may be direct or indirect and may affect the muscles themselves, the nerves supplying them, or both. The injury may involve the orbital structures, or be intracranial. Instances have been cited in which ocular palsies have followed orbital injury. Bullet wounds, or perforations of the orbit by sharp instruments and foreign bodies, direct blows upon the eyeball and the orbital contents and intranasal operations are all capable of producing paralysis of one or more ocular muscles. The relation of cause and effect is patent and not difficult to determine. No less traumatic in origin, but more obscure, are the instances of ocular palsy and proptosis of the eyeball following spontaneous subperiosteal hemorrhage, which may occur during a paroxysm in whooping-cough, or in scurvy, especially in young children. In these diseases, subperiosteal hemorrhage is common.

Intracranial trauma may be direct or indirect. Fracture of the base of the skull is usually accompanied by sixth nerve palsy. The palsy may be due directly to the fracture, to compression by exudate thrown out, or to hemorrhage. In a majority of instances, diplopia is present shortly after the fracture occurs, and disappears during convalescence. When the sixth nerve palsy appears sometime after the injury, it is apt to be the result of pressure from exudate and blood. In some instances, the nerve is torn at the time of the injury. In the author's experience, sixth nerve palsy after a head injury is strong presumptive evidence of a basal fracture even though the Roentgen-ray plate may fail to disclose a line of fracture. The symptom of diplopia should be routinely sought in all head injuries even though the patient may not complain of its presence.

Aside from direct injury in a basal fracture, or from blood extravasation from such injury, a large meningeal hemorrhage from a blow with or without a depressed fracture of the skull, may be followed by a sixth nerve palsy from increased intracranial pressure alone. The exposed course of this nerve after it emerges from the brain and before it leaves the skull, renders it peculiarly liable to suffer from a remote injury to the skull or from blood extravasation. It may, therefore, be present in fractures of the vault of the skull or simply from blood extravasation following a blow on the top of the head.

The other ocular nerves are not so apt to be paralyzed in skull trauma, although severe injury and blood extravasation may involve the fourth and branches of the third nerve as well.

Inflammatory and Vascular Causes.—Probably the greatest number of ocular palsies are caused by inflammatory and associated vascular disease. Of these processes, syphilis heads the list. For the most part syphilitic palsies are of intracranial origin and usually are due to syphilitic meningitis. The third nerve or one of its branches is more frequently involved than the fourth or sixth. As an isolated palsy due to syphilis, ptosis is most common. Complete third nerve palsy is usually of trunk origin, the nerve being compressed in an inflammatory mass. Gummatous growths or inflammations may also occur in the deeper structures of the brain and involve the nuclei. It is probable that isolated ptosis may be of nuclear origin or fascicular, although there is evidence for and against the same. The fourth and sixth may also be paralyzed by the same process alone or combined with the other nerves of the eye. In the orbit, syphilitic periostitis, which usually involves the upper and inner orbital rim, may be associated with palsy of the superior oblique. Chronic dacryocystitis accompanied by bone necrosis may cause involvement of the inferior oblique. The relation of internal ophthalmoplegia to lues as a cause is well known.

Basal tuberculous meningitis is a well-established factor in many cases of ocular palsy. In fact, any form of basal meningitis may act as a cause.

Paralysis due to otogenic pathology is perhaps less frequent than that of nasal origin, but quite common. The Gradenigo syndrome of abducens palsy, associated with middle-ear disease and Gasserian ganglion pain, is of more frequent occurrence than one would be led to believe from a review of the literature. The most complete review and analysis of this syndrome is that of William H. Sears.[1]

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[1] Otogenic Paralysis of the Abducens, Trans. Am. Laryngol., Rhinol. and Otol. Soc., 1925.

The sixth nerve may be involved homolaterally, contralaterally, or bilaterally. The former is usual; crossed paralysis and bilateral involvement are rare. The onset is rather late, appearing from ten to thirty days, but occasionally as late as two months, after the otitis media is noted. Factors which seem to account for sixth nerve palsy are direct extension of the tympanic pus or the toxic process "inwardly through the pyramid to the apex." This is especially found when the bone is extensively pneumatized. Indirect routes are many. The process may follow along veinous channels to the petrosal sinuses and jugular bulb by thrombophlebitis, or along any of the numerous tracts which carry nerves and vessels, along the lymphatic stream, by a pachymeningitis or leptomeningitis, or from an extradural abscess. Edema of the structures surrounding Dorello's canal, with the resulting pressure on the sixth nerve causes the paralysis. To these avenues of attack, Cushing[1] adds the possibility of pressure by the basilar artery in the presence of serous meningitis.

Occasional contralateral and bilateral paralysis by pressure of the basilar artery in the presence of serous meningitis is explained, by coincident contralateral sphenoidal sinus disease, by direct contact with the sixth nerve and by increased intracranial pressure, which may follow ligation of the internal jugular vein.

For a more complete study of this interesting syndrome, the reader is referred to Sears'. article.

Inflammatory processes in the orbit, which lead to ocular nerve palsy, are mostly of sinus origin. Attention has been called to the relation of the orbital contents, and particularly the muscle cone, to the sphenoids, ethmoids, and the frontals. A diseased antrum is possibly less likely to lead to paralysis than are the other sinuses. It is worth recording that many acute palsies of sinus origin may be due to acute sinus disease without bone necrosis, and that prompt opening, particularly of the sphenoid, followed by irrigation, in many instances brings about immediate recovery.

The author has observed a number of cases of isolated ocular muscle palsies due to this cause. One case, in particular, will serve to illustrate the group. A young man, aged twenty-two years, suddenly developed paralysis of the right internal rectus. All laboratory and clinical studies proved to be negative with, however, a suspicion of sphenoidal involvement. Opening up of the sphenoid and irrigation were followed within twelve hours by complete restoration of function of the paralyzed muscle.

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[1] Brain, vol. 33, 204.

A serious cause, from a prognostic standpoint, is that of cavernous sinus thrombosis. The rapidity of onset, the completeness of the ophthalmoplegia, associated with proptosis and the rapid spread to the other eye, are a well-known sequence of events, which lead to a fatal issue. On the other hand, one occasionally meets with a toxemic form, similar in character, in onset, and development, but which results in recovery. The author observed a case of this character several years ago with this happy result. A priest, aged thirty-five years, suddenly developed proptosis, first of one eye, then of the other, with complete ophthalmoplegia, an irregular temperature, accompanied by chills and slight delirium. A blood culture showed the presence of the pneumococcus. A small patch of central pneumonia was discovered. Under intravenous medication, recovery gradually took place, with complete restoration of function of all the ocular muscles.

In this group should be noted recurrent third nerve palsy, also called "ophthalmoplegic migraine." Although the third nerve, or one or more of its branches is usually affected, paresis of the fourth and sixth nerves has been noted either singly or associated with third nerve involvement. The onset with pain followed by ocular palsy, its spontaneous recovery. and its periodicity are its chief characteristics. Its exact etiology is unknown. Syphilis, tuberculosis, malaria, pachymeningitis, hysteria have all been assigned as causes, but many cases have been observed in which each of these conditions has been ruled out. It is probable that it bears some definite relation to the sensory forms of migraine in its etiology. Just what the underlying cause is, remains to be determined. Vasomotor disturbance may account for the sensory form, but it does not satisfactorily explain the duration of the symptoms of the ophthalmoplegic variety.

Ocular palsies are observed in nephritis and in diabetes. Vascular disease, hemorrhagic and edematous conditions are probably responsible. Influenza, acute hemorrhagic encephalitis, lethargic encephalitis are all well-known causes of ocular muscle palsies, for the most part infectious in origin. Other causes which should be included in this group are multiple sclerosis, acute bulbar paralysis, brain abscess, acute anterior poliomyelitis, paralytic dementia, vascular diseases, including arteriosclerosis, aneurysm, thrombosis, embolism and rupture, myasthenia gravis, and spinal anesthesia. The last cause should be specially stressed. Paralysis of the external rectus has been observed a number of times. It comes on rather remotely two weeks after the spinal anesthesia. Recovery, as a rule, is assured.

Neoplastic Causes.—Tumors of the brain are often accompanied by partial or complete ophthalmoplegia. They may produce a paralysis by direct pressure or by increased intracranial pressure. Syphilitic gummata, if included in this group, are responsible for many cases. As a rule, action is direct, the nerves or nerve centers being imbedded in the gummatous masses. Tumors in remote parts of the brain may cause sixth nerve paralysis by increased intracranial pressure. This is particularly true of tumor of the posterior fossa which are apt to cause stretching of the arteries around the nerve stem. Cushing has observed "in cases of extreme constriction, the nerves may be found degenerated, and replaced by scar tissue at the point of constriction." He further points out that "convergent squint may disappear after the successful removal of a cerebellar tumor."

Sixth nerve palsy due to brain tumor is not of much localizing value, therefore, because it usually is caused by increased intracranial pressure. Of more localizing value are the single or multiple palsies which occur from syphilitic gummata.

Tumors of the orbit are apt to lead to more or less complete ophthalmoplegia because of the compression of the retrobulbar structures. In the early stages, however, of a new growth of the orbit, the paralysis may at first be confined to a single muscle, others becoming involved as the growth develops.

Toxic Causes.—Although chronic nephritis and diabetes might be included in the toxic group, they more correctly belong to the vascular and inflammatory causes. The acute infectious fevers which are apt to lead to paralytic squint are diphtheria, influenza, typhoid fever, and malaria, although isolated cases have been observed after any of the infectious diseases of childhood.

Among the intoxicants are lead, alcohol and other drugs, ptomain poisoning, spinal anesthesia, etc.

Symtomatology.—The symptoms of paralytic squint may be discussed: (1) From the standpoint of signs and symptoms upon which the diagnosis of an ocular palsy is based; and (2) from the standpoint of correlated symptoms in order to arrive at an opinion as to the character and location of the lesion. The first is essential in the diagnosis and isolation of the palsy, and the second is necessary from the viewpoint of prognosis and treatment.

Primary Deviation.—If one of the lateral rotators is paralyzed, squint is evident upon even a casual inspection. Inasmuch as the antagonist of the paralyzed muscle is intact, the eye will deviate toward the non-paralyzed lateral rotator, or away from the paralyzed muscle. The degree of deviation from "eye front" will be in direct proportion to the completeness of the palsy. If the palsy is incomplete, there may be no appreciable deviation of the eye from parallelism by ordinary inspection. If, however, the patient is asked to fix at 6 meters, and the position of the corneal reflection of a small electric light, held directly in front of the patient, is noted, even a moderate paresis may be detected. The corneal reflections will not occupy the same relative positions with reference to the pupillary centers. The presence of a positive or negative angle kappa, must of necessity be determined if the paresis is moderate in degree. If the right internal rectus, for example, is paralyzed, the corneal reflex in the right eye will be found toward the inner edge of the pupil, because the intact external rectus produces a certain amount of external rotation.

When a vertically acting muscle is paralyzed, ordinary inspection may not disclose much if any vertical deviation. This is due to the fact that there are two muscles active in each cardinal vertical movement, and the displacement is not so marked even in complete palsies. The test by means of the corneal images from an electric light must usually be employed to detect vertical deviation.

The cover test is always of value in determining the presence of paralytic deviation, either in lateral or in vertical rotators. In moderate degrees of squint, however, it does not definitely differentiate paresis from a phoria. As the cover is passed from one eye to the other, and the position of the paralyzed eye is carefully noted under cover, the deviation becomes manifest. One may also observe the eye, as it is uncovered to fix on a distant object and its movement may be noted. Its behavior behind the cover is quite conclusive. Furthermore, as the same test is used in the determining of secondary deviation, by observing the behavior of the eyes behind the cover, one obtains a better idea of the relation of primary to secondary deviation.

Secondary Deviation.—Under normal conditions, when a patient is asked to fix a distant object, he will fix with the non-paralytic eye, the eye with the paralyzed muscle undergoing deviation. This is called *primary deviation*. If the normal eye is covered and the patient is asked to fix a distant object with the paralytic eye, the normal eye is observed to assume the position which the affected eye assumed in primary deviation. This assumed position of the normal eye is known as *secondary deviation*. If one carefully notes the position of each eye under cover in these experiments, it will be observed furthermore that the primary deviation of the paralyzed eye is less than the secondary deviation of the normal eye. The reason for the difference in the amount of deviation is as follows: The innervation required of the non-paralyzed eye to fix when the paretic eye is covered, is a normal innervation, the paralytic eye simply failing to follow in this conjugate movement because the yoke muscle is paralyzed. In secondary deviation, when the paralytic eye fixes, in primary position or "eyes front," a greater effort is required to hold this eye in position because of the paralytic muscle, and this same excess of energy goes into the yoke muscle of the non-paralytic eye, causing it to deviate excessively. For example, if the right internal rectus is paralyzed, in the cover test or without the cover test, the right eye will deviate to the right while the left eye fixes. In secondary deviation, if the cover is applied to the left or normal eye, and the right eye is forced into fixation, it will be noted that the left eye will be turned to the left, and to a greater extent than was the primary deviation of the right. Strictly speaking, these movements to the right and left are not wholly brought about by the conjugate muscles, but by the synergists of each group; inasmuch as the movements are weakened by a paralysis of the chief muscle in the group of synergists, we speak of the action as though it were limited to a single muscle, the conjugate of the fellow eye.

Ordinarily, as one inspects a patient suffering from paralytic squint, he observes the primary deviation. It so happens in a few instances, however, that secondary deviation may be present and not primary. This takes place when the non-paralytic eye is amblyopic. In such instances, the paralytic eye possessing the better vision will assume the role of fixation, and secondary deviation will be present in the non-paralyzed eye. This fact can be determined by a study of the limitation of movement of each eye and by the fields of fixation. The phenomenon is not often observed without the cover test, but it is of sufficient importance to confuse the student in correlating his symptoms if he is not on the look-out for its occurrence. If cases are studied, as they should be, in a routine manner, the presence of amblyopia or of much reduced vision in one eye will place the student upon his guard for the detection of this possibility.

Limitation of Movement.—More conspicuous than primary deviation is the varying degree of the angle of squint. This is due directly to the paralytic muscle. When the eyes are turned in a direction in which the paralyzed muscle is not an important factor, there is little if any noticeable deviation. When, however, an attempt is made by the patient to look in the direction of the paralyzed muscle, the afflicted eye responds but feebly, and finally comes to a standstill, while the fellow eye makes its complete rotation. Limitation of movement, therefore, is an important diagnostic factor in the study of paralytic squint. For example, when a patient suffering from paralysis of the left external rectus attempts to look to the left (*i. e.*, in the direction of action of the paralytic muscle), the left eye may fail to move beyond the center, or at most may only be rotated outward 15 to 20 degrees. The right eye, however, makes its full rotation because it responds normally to the bilateral innervation to the left. Limitation of movement is most noticeable in paralysis of one of the lateral muscles, to a less degree in paralysis of the vertical recti, and least of all in paralysis of the obliques. If an elevator or depressor muscle is under study, the limitation should be noted not only in the cardinal directions, but in the oblique directions as well. One must clearly bear in mind the normal action from primary position and the maximum vertical pull when the eyes are turned in the opposite direction. The right superior rectus, for example, from eyes front, normally rotates the eyeball up and in, but the maximum vertical elevation of the eye is obtained by this muscle when the eye is rotated to the right—the right superior rectus being a right-hand elevator. In paralysis of the right superior rectus, therefore, there will be limitation of movement up and in, but greater up and out. According to E. and M. Landolt,[1] the up-and-in movement will be less than the up-and-out movement. In the author's experience, the greatest limitation is in the direction of the normal maximum vertical pull of the muscle. This corresponds to the field studies of Landolt, as shown in Fig. 86, which is an anatomical field of fixation similar to that of the Landolts. The limitation in this field is up and in, or in the direction of the maximum pull of the inferior oblique. The position of the paralyzed eye will be influenced by the action of the five ocular muscles left intact. In this paralysis, therefore, the eye will be down and in. Although the maximum up and in movement is brought about by the inferior oblique, this movement is set under way by the superior rectus.

An up-and-out limitation will also be noted. The extreme up-and-out rotation is accomplished by the superior rectus, but it is set under way by the inferior oblique. When the paralysis of a given muscle is only partial, there may be some difficulty in detecting any limitation of movement. Under these circumstances, a symptom which is very characteristic, as a rule, is of value. Instead of a noticeable restriction in the rotation of the eye, the observer will observe, when the eye reaches the extreme end of the rotation, there will be coarse nystagmoid or jerky movements of the afflicted eye, while the opposite eye remains in position of extreme rotation. This symptom is quite characteristic and indicates that the muscle, although not completely paralyzed, cannot sustain its full innervational stimulation. When a complete palsy, on the other hand, shows evidence of improvement, this jerky nystagmoid movement is one of the earliest signs of recovery.

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[1] Defective Ocular Movements and Their Diagnosis, page 44.

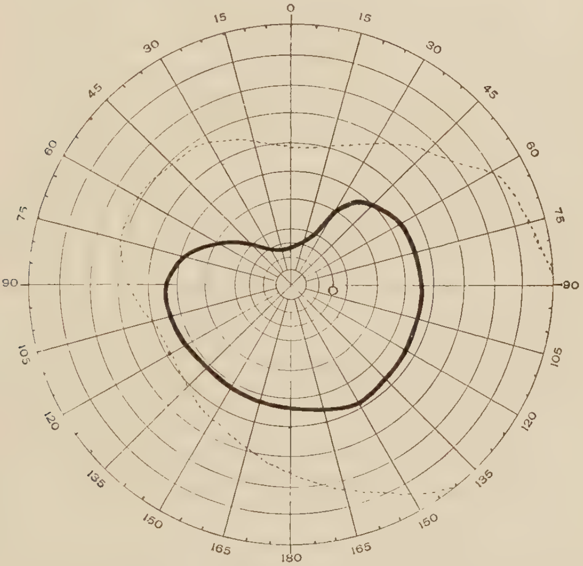


Fig. 86.—Anatomical field of fixation of a *left* eye affected with paresis of *the inferior oblique.*

If carefully observed, limitation of movement is quite illuminating from a diagnostic standpoint. It serves as a third differentiating sign between concomitant and paralytic squint, the first, as already pointed out, being a variation in the angle of squint in various parts of the field of fixation, and the second, the excess of the secondary deviation over the primary which does not obtain in concomitant squint.

If there is doubt in the mind of the observer that a limitation of movement exists, a more graphic method of study may be employed, namely a study of the field of macular fixation. This study is made on the perimeter in one of two ways, either by a subjective or by an objective method.

Perimetric Measurements.—In the former, or subjective method, the patient is seated before a perimeter, as though the visual fields were to be taken. One eye is blindfolded and care is observed that the patient's position is correct for the eye under observaton. The test object consists of a card on which is printed a word in type sufficiently large that the patient can read it distinctly. If the patient is presbyopic, the type must necessarily be larger. The test card is now moved along the arm of the perimeter in a horizontal direction, and the patient is instructed to follow the card with his eye, *without turning the head,* until the type blurs. This will indicate the extreme point of rotation in that direction. Eight points of study at least are essential, the four cardinal directions and the oblique meridians between the cardinal directions. When in the field of action of the paralytic muscle, an even greater number of points of study should be made so as to obtain a more accurate idea of the limitation. Care should always be observed that the letter in the most extreme periphery is the real test to be depended on, otherwise an error of 5 or even 10 degrees may be made in the measurements.

The results obtained may be charted on an ordinary perimetric chart, as illustrated in Fig. 87, and the points may be connected by lines. The finished chart will give one a graphic representation of the limits of rotations.

The objective method which is less accurate is as follows: Instead of using the test object as described above, the patient is asked to follow a small electric light which is moved along the arc of the perimeter until the extreme limit of rotation is reached. The operator standing back of the perimeter right over the light, notes when the corneal reflex is perfectly centered in the pupil. The degree of rotation is read off from the perimeter and recorded. The study of the other parts of the field of rotation are made in a similar manner. Results by this method are not so accurate as by the subjective method just described.

The method of recording the field of rotation, as practised by the author, saves much time and is quite as satisfactory as the perimetric chart method Fig. 36 p. 74.

A careful comparison of the fixation fields of the two eyes will furnish interesting and accurate information. For example, let the student carefully observe a field of rotation of paralysis of the left external rectus. He will note that the excursion in the horizontal meridian is less than the excursion up and out, and down and out. The intact vertically acting muscles carry the rotation further out and up, and out and down than the external rectus can carry the eye in a horizontal direction. This indentation in the horizontal meridian differs materially from the curve in rotation when present in a concomitant lateral squint (Fig. 78). In the latter, the horizontal limit is greater than the lateral limit up and out, and down and out. This difference in the fields of rotation offers a fourth point in the differentiation between a paralytic and concomitant squint.

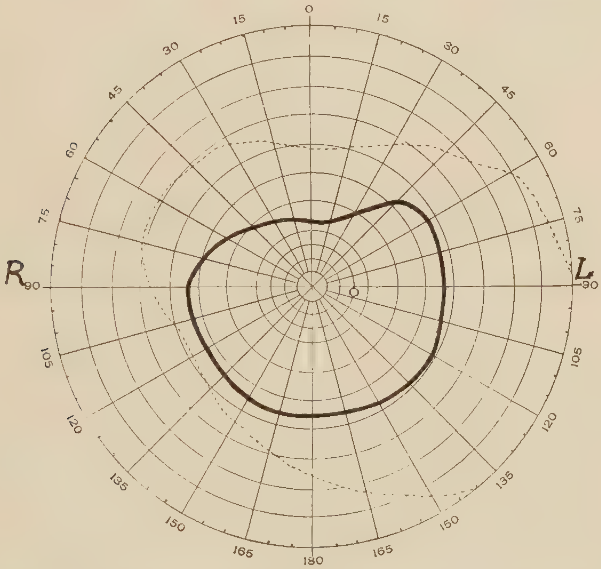


Fig. 87.—Method of recording the fields of fixation (anatomical).

Tropometric Measurements.—The rotating power of the eye may be measured on Stevens' tropometer. This instrument is well adapted to measure the rotations in the cardinal directions, but does not lend itself to oblique rotations which are quite as important. Furthermore, the instrument is rather cumbersome, and adds another instrument to one's equipment which is unnecessary if the same studies are made on the perimeter. In the author's experience, the perimetric method is quite as accurate as the tropometric method.

False Projection and False Orientation.—In paralysis of one or more ocular muscles, the patient is unable to accurately locate an object in space, because the object does not fall upon corresponding retinal points. The object fixed by the macula of the normal eye, is to the inner or outer side, or above or below the macula of the paralyzed eye, as the case may be. This gives rise to an uncertainty in the gait and in the execution of ordinarily simple acts, which, however, demand careful coördination of both eyes in binocular vision. The patient tends to locate the false image by the ordinary amount of rotation required under *normal* conditions to bring the macula in place in binocular single vision. For example, if the left eye is turned in 20 degrees because of paralysis of the external rectus muscle, the image of the point fixed will fall 20 degrees to the inside of the left macula.

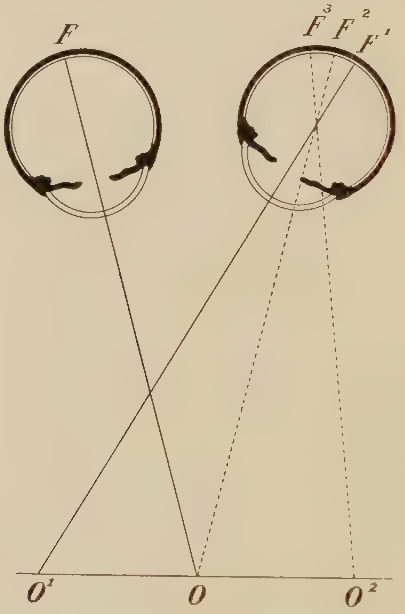


Fig. 88.—False projection in paralysis of the left external rectus.

In Fig. 88, F represents the macula of the right eye and F^1 that of the left eye. The object O falls upon F in the right eye and upon F^2 in the left eye, 20 degrees to the inner side of the left macula F^1. In normal conditions, there would be an impulse to rotate F^1 20 degrees inward, equivalent to an outward rotation of the cornea 20 degrees to bring O^1 to O, or F^1 to F^2. In a state of paralysis, the same condition obtains but the premise is false. O falls upon F^2 instead of F^1. The same mental act prevails, but the projection of O is to the position of O^2 because O falls upon F^2 which is 20 degrees to the inner side of F^1 and the projection is as though F^2 were moved to F^3, which is 20 degrees to the inner side of F^2. The projection is a false mental projection. The false projection is in the field of action of the paralyzed muscle.

When a patient fixes an object within his reach, and is then asked to point to the object, the normal or fixing eye now being occluded, he will point beyond the object in the direction of the paralyzed muscle. He "pass points," so to speak. If he is asked to walk to a distant object under the same conditions, he will also tend to walk too far in the direction of the paralyzed muscle.

The degree of false projection will depend upon the degree of paralysis and upon the length of time the paralysis has existed. After a time, patients learn to correct the error, and by a little practise, they can point to or walk towards an object with a fair degree of accuracy.

Another factor probably enters into false orientation and false projection. The amount of innervation required to bring an eye, suffering from a paralyzed muscle, into fixation may influence the misinterpretation of the location of an object.

Landolt's apparatus may be used to accurately measure the degree of false projection. For lateral palsies, the apparatus consists of a horizontal board attached to a wall by hinges. The horizontal has an indentation for the patient's neck. Under the board is arranged a tangential scale to the right and left of the center. With the non-paralyzed eye closed, the patient is asked to point underneath the board, to the vertical line in the center. The amount of error on the tangent can be read off in degrees of false projection. The value of the instrument lies in the inability of the patient to see just where his finger points in the test. For clinical work, the apparatus is not essential, as the simple "touch test" of von Graefe is sufficient for all practical purposes.

Diplopia.—The most distressing symptom of which the patient complains, and at the same time a most valuable guide from a diagnostic standpoint, is double vision or diplopia. It is this symptom and the discomfort caused by it that leads the patient to consult the oculist. Essentially it is a symptom of paralytic squint, and is rarely observed in the concomitant variety. With the deviation of the eyeball from parallelism with its fellow eye, caused by the action of the antagonists against a weakened or totally paralytic muscle, or muscles, there appears in the patient's field double images of objects fixed by the eyes. The true and false images are separated, and the fusion faculty cannot operate satisfactorily, because of the paralytic muscle, in the interests of single binocular vision. Which is the true image and which the so-called false image cannot always be determined by the patient in the first days of diplopia. In other instances careful analyses are made by the patient and he can furnish most valuable assistance to the surgeon in arriving at definite conclusions as to the affected muscle or muscles.

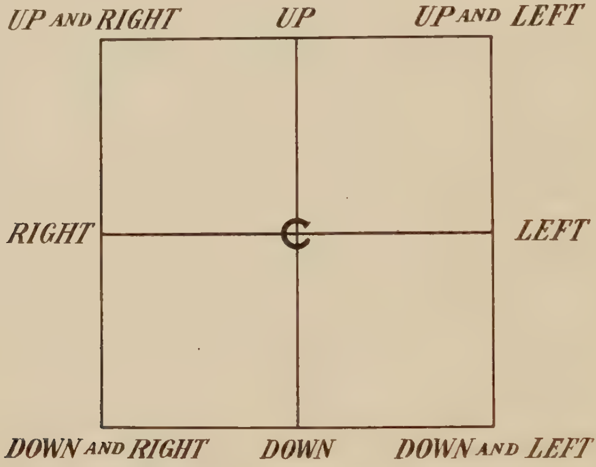


FIG. 89.—Points to be studied in the diplopia field, beginning at *C*, or center.

Method of Studying Diplopia.—Studies of diplopia are greatly facilitated by the use of a red glass over one of the eyes, so as to enable the patient to clearly distinguish between the images of the two eyes, and to determine the relative position which the false image bears to the true one. The following phases need careful analysis in determining which muscle is paralyzed: (1) The relative position of the two images when the eyes are directed forward; (2) the cardinal direction in which the separation is greatest; (3) in vertical diplopia, the maximum vertical separation on looking to the right or to the left, in addition to the cardinal directions of the separation; (4) the obliquity of the false image; (5) in a certain proportion of cases, the direction of maximum lateral separation; (6) the question of the crossed or uncrossed character of the diplopia. For reasons which will be fully set forth, the fifth and sixth phases of the subject are of least importance from the standpoint of annoyance to the patient and from the standpoint of trustworthy evidence in diagnosis.

Vertical separation is the symptom which causes most distress to a majority of patients, while the obliquity of the image in a limited number of cases offers the greatest discomfort. Lateral separation is least distressing.

A study of diplopia must proceed in an orderly and methodical manner, and if so approached will rarely offer difficulty even to the beginner. The investigation should be conducted in a dark room if possible, or at all events in a room in which the light is much reduced. A red glass should be held by the patient in front of one eye. It matters little which eye is selected. The examiner takes his place 1 or 2 meters in front of the patient. For this study, a lighted candle may be employed, or the light of a luminous ophthalmoscope or, better than either of these, a rod of light 3 or 4 inches long will be of special value in determining the tilting of the false image.

The light is held directly in front of the patient on a line with his vision. If double images are present the fact is noted, together with their relative positions. If diplopia is absent in this part of the field, or if present, the operator proceeds to the second step of investigation, namely, to pass the light up and down from the center and to right and left of the center, to determine in which of the four cardinal directions the diplopia appears and the separation increases. It is an established fact that diplopia increases in the direction of the normal field of operation of the paralyzed muscle. For example, the external recti muscles move the eyes templeward; therefore, in paralysis of an externus the eye is turned in by the antagonists, and the diplopia increases toward the temporal field, or in the direction of the muscle. A diplopia, therefore, which is developed or increased on turning the eyes to the left, would indicate a paresis or paralysis of a left-hand turner, *i. e.*, either the left external rectus or the right internal rectus. In lateral diplopia a third step is necessary. One must determine which is the true and which is the false image, and to which eye the false image belongs. The first of these tests is a known quantity. By false projection, the so-called false image is always the distal image. If the eye is rotated in, the false image is projected outward, and the outward projection is in direct proportion to the inward rotation. It is established, therefore, that the distal image belongs to the paralytic eye, so to speak. It only remains to determine by means of the cover test or, better still, by means of the red glass, to which eye the false image belongs. In diplopia increasing to the left, if the red glass is placed before the left eye, and the red image is to the left of the yellow light, the false image must belong to the left eye. The left external rectus, therefore, is readily selected as the paretic muscle.

The beginner may be confused in lateral diplopia by the fact that there may be two objects in the upper and lower left-hand quadrants as well as in the left horizontal meridian (Plate, IV). A careful analysis, however, will reveal the fact that it is the lateral separation which is greatest, and that the vertical separation is slight or almost absent in the upper and lower quadrants. The cardinal direction of the diplopia is to the left, and it, therefore, impeaches a left-hand horizontal turner.

When the upper or lower field shows the greatest tendency to separation the analysis must go a step further. Diplopia in the upper field throws suspicion upon the four elevator muscles—the two superior recti and the inferior obliques—because these muscles are elevators or upward rotators. The same is true of the lower field. If the vertical separation of the images increases in the lower field, one of the depressor group is involved. The patient is now asked, as the light is passed up, and to the right, and up and to the left, does the *vertical* separation increase on looking to the left or to the right? If to the left and up, a left-hand elevator is involved as the *diplopia* increases in the direction in which the paralyzed muscle acts, *ad maximum*, as an elevator. "The image is as the muscle was," as Maddox has aptly put it. There are two left-hand elevators, the *left* superior rectus and the *right* inferior oblique. By means of the red glass, the eye to which the false image belongs is easily selected. If the red glass is placed before the left eye, and the red image is higher than the yellow light, the left eye is the eye to which the paralytic muscle belongs—paralysis of the left superior rectus.

The student asks "Does not the left superior rectus move the eye up and in?" Yes! Why then shall it be regarded as a left-hand elevator? The superior rectus primarily turns the eye up about its horizontal axis, but, secondarily, it turns the eye in, and rotates the upper end of the vertical meridian of the cornea in. When, however, the eye is rotated outward 23 degrees or more, *i. e.*, when the muscle plane coincides with the antero-posterior axis of the globe, there can be but one direction of movement, and that will be straight up. Theoretically there will now be no lateral pull, and no torsional action, but all the kinetic energy of the muscular contraction will be spent in a direct upward pull. Because, therefore, the left superior rectus moves the eye up *ad maximum* when the eye is turned outward or to the left, it has been very appropriately called (Duane) a left-hand elevator. If this is its function in health the vertical diplopia must necessarily increase when the patient attempts to look in this direction when the muscle is paralyzed. The false image is in a direction opposite to the deviation assumed by the eye in paralysis. It will be helpful to the student to know that his diagnosis of the muscle involved is correct.

In order to do this in a diagrammatic manner, observe that in Fig. 90 *LSR* is the paralytic muscle. Two muscles with a downward pull oppose one with an upward pull. The strength of *LIR* and *LSO* is greater than that of *LIO*, therefore, the eye will be displaced downward. Two muscles, *LIO* and *LSO*, have an outward pull against *LIR*, which rotates toward the nose. Therefore, the eye will be displaced outward and downward. If the eye is down and out the false image must be up and in, *i. e.*, in the direction which the left superior rectus normally acts from, "eye front."

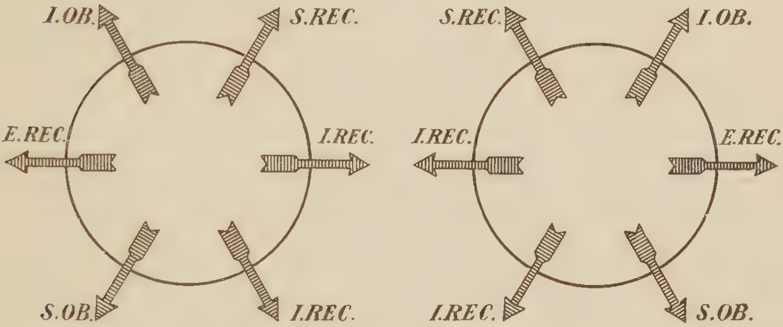


Fig. 90.—Diagram showing the normal action of each muscle from "eye front."

An intelligent patient, however, will volunteer the information that the false image is not only up and in, but that the upper end of the false image leans toward the nose. If this is correct the vertical meridian of the cornea must lean outward toward the temple. That this is correct can be readily verified by again referring to Fig. 90. The two superior muscles, the superior rectus and the superior oblique, rotate the upper end of the vertical meridian of the cornea in. The two inferior muscles rotate the upper end of the corneal meridian out. If the superior rectus is paralyzed there will be but one intortor of the corneal meridian, namely, the superior oblique. The two extortors of the cornea, however, are intact, namely, the inferior rectus and the inferior oblique. The preponderance of force must necessarily be in the direction of extortion.

The eye, therefore, is not only displaced down and out, but the vertical corneal meridian will be in a position of extortion. The false image will lean in the opposite direction and will be found to be up and in and intorted.

Each of the vertically acting muscles should be selected in turn on the diagram, as in the case of the left superior rectus, and thus should be carefully studied. Memory methods may stick for a time, but a process of reasoning will enable the student to make more correct diagnoses.

In following the chart (Fig. 90), to locate the position of the false image, the chief difficulty lies in the vertical separation in reference to the right- and left-hand vertical elevators. The student must thoroughly grasp the fact that vertically acting muscles have a primary and a secondary function, and that either function may be increased at the expense of the other. The vertical recti may become simple elevators or depressors if the eyeball is rotated outward so as to give a straight pull up or down. As a rule, there is also some lateral separation of the two images. Instead, therefore, of a simple vertical separation, the lateral separation should also be studied. In the case of the left superior rectus the lateral separation and the obliquity of the false image will be found to increase when the testing light is moved up and toward the normal side. The reasons for this are apparent. When the left eye, for example, is turned toward the healthy side, the superior rectus becomes more of an internal rotator and an intorter, and less of an elevator. When the muscle is paralyzed and the gaze is directed in this part of its normal field of action, according to the laws of paralyzed muscles, the false image will naturally separate further in a horizontal direction from the true and the increasing preponderance of the inferior or extorting antagonists will shift the vertical meridian of the cornea further out, and the inward leaning of the false image will be proportionally greater.

For purposes of diagnosis the lateral separation is not essential. The increase in the obliquity of the false image, however, is of much practical importance, as will be indicated in certain cases by the great tendency of the patient to tilt the head to the right or the left shoulder, as the case may be, in order to correct the tilting of the image. This symptom becomes of special importance in the application of surgical measures to correct this defect in those patients who find the leaning of the false image particularly annoying.

The crossed or uncrossed character of the diplopia has received considerable attention by some authors and less by others. Paralysis of the vertical recti, as a rule, produces crossed diplopia, and that of the obliques homonymous displacement. In this they correspond normally to paralysis of the internal and external recti, respectively. There is this difference: The laterally operating internal and external recti have but one action, that of lateral rotation, whereas the vertical muscles are only secondarily internal and external rotators. In paralysis of the lateral rotators diplopia is so marked that a preëxisting esophoria or exophoria will have little or no perceptible effect upon the separation of the images. In the case of the vertical muscles, lateral separation is slight, as the lateral movements of these muscles in health are only secondary. If the left superior rectus is paralyzed there should be a slight crossed diplopia. If, however, there was a preëxisting esophoria it might neutralize the crossing of the images, because in esophoria there is a tendency to uncrossed lateral separation. In fact, under such conditions, the lateral diplopia, if any, might be uncrossed. The same may be the result in paralysis of an oblique with a preëxisting exophoria. In actual experience, therefore, the uncrossed or crossed character of the diplopia cannot be depended upon to follow the rule, and the student will do well to ignore this phase of diplopia when the superior muscles are under investigation.

In most instances diplopia is of greatest annoyance at the beginning of the palsy. It may continue unabated if the palsy is permanent, although a majority of patients quickly learn how to diminish the separation by compensatory face turnings and head tiltings. In those instances in which recovery follows in the course of a few weeks or months the discomfort grows less and finally disappears. Lateral diplopia is less troublesome because the separation occurs mostly in the horizontal meridian, and the patient promptly learns how to minimize lateral separation of the images. In paralysis of the vertical muscles the movements are more involved by lateral and torsional complications, and the correction of the same by vicarious rotations of the head is not so easily accomplished.

Torsion, in a limited number of cases, gives rise to the symptom which usually is described as "swimming," and may be accompanied by nausea and vomiting in paroxysms. In the writer's experience the cases which are most intractable, and in whom a secondary neurasthenic condition supervenes, torsion of the false image is the underlying condition, perhaps even more pronounced than vertical displacement.

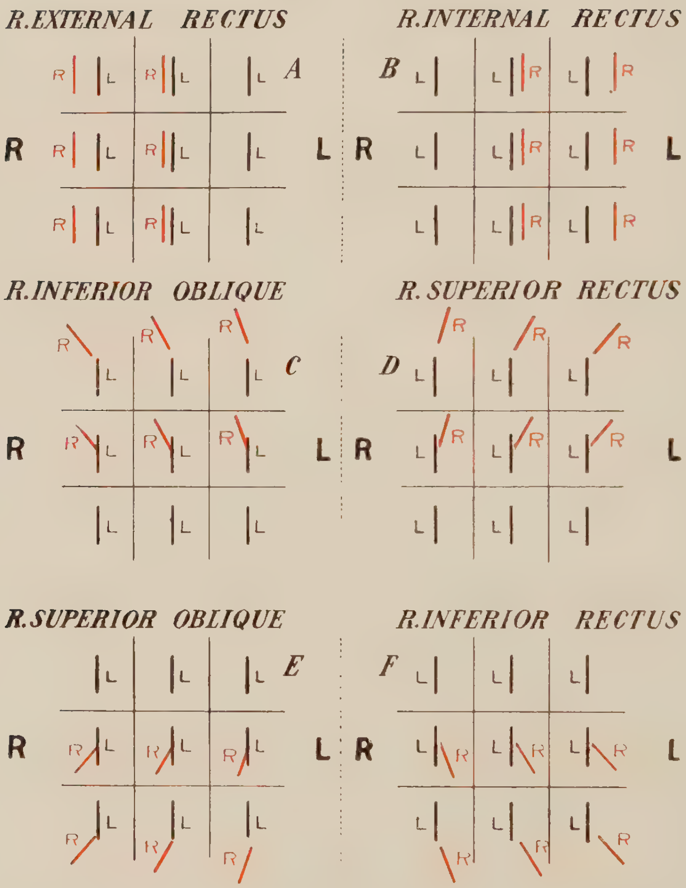
Unfortunately for the adult who suffers from incurable paralytic squint, macular vision does not, as a rule, suffer deterioration. In this respect it is unlike the concomitant type of squint which occurs, as a rule, in the child and in whom amblyopia of a profound character develops in many instances. In cases of concomitant squint which develop late in life, and macular vision is preserved, the patient, as a rule, promptly learns to suppress one image, and is not annoyed by diplopia. The paralytic squinter learns less readily to suppress the image and, therefore, largely overcomes the diplopia by "vicarious rotations of the head."

In the following diagrams are recorded the diplopia fields of each eye. In these diagrams the lateral separations are recorded according to rule. No stress, however, is laid upon the lateral separation of the two images in the vertical palsies, and no attempt is made to faithfully record the exact leaning of the images. In Plate IV are recorded the images in paralysis of each of the muscles of the right eye. In Plate V are recorded the images in paralysis of each of the muscles of the left eye.

Vertigo.—Vertigo, dizziness, "swimming," are terms which patients use in explaining the subjective sensations experienced in ocular palsies. They are the direct result of double vision or of diplopia and false projection. They are present from the beginning and oftentimes grow worse as the patients run into months and years of uncorrected or uncorrectable paralytic squint. These symptoms naturally are mild or severe: (1) In direct proportion to the amount of displacement of the images; (2) in direct relation to the type of palsy, *i. e.*, as to whether it is a lateral rotator which is involved or an elevator or depressor; (3) in direct relation to the normal status of the individual's nervous balance. Lateral separation is less likely to give rise to the marked subjective symptoms referred to in this paragraph, other things being equal, than a vertical separation, because of the complicated character of diplopia in the latter group.

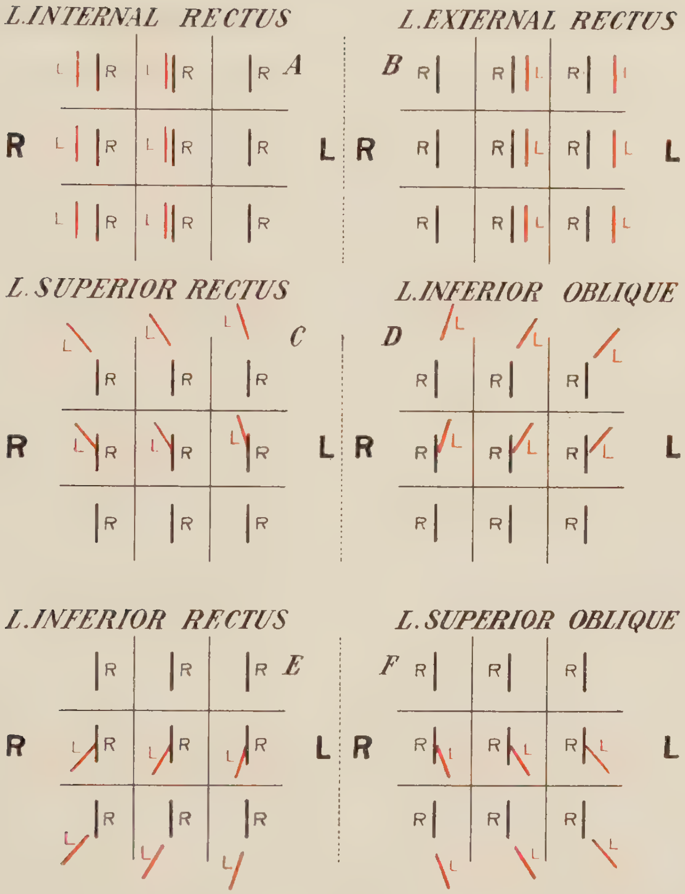
The severity of the symptoms, however, is more dependent upon the state of the nervous equilibrium of the patient than upon the character of the diplopia. In the unstable a most profound neurasthenic state may be the ultimate outcome of an incurable palsy, if one eye is not occluded. The writer has never observed a more profound case of helpless and almost hopeless neurasthenia, which for a time seemed to threaten a totally and permanently unbalanced mind, than in a badly neglected case of ocular palsy of fifteen years' standing. The patient had a persistent torticollis, chronic in character, which finally involved the entire shoulder girdle as the result of a voluntary effort on the part of the patient to correct the diplopia

PLATE IV



Images in Paralysis of Each of the Muscles of the Right Eye.

PLATE V



Images in Paralysis of Each of the Muscles of the Left Eye.

by vicarious rotations of the head. She was unable to walk, and could not even support her head in the sitting posture. She was practically bed-ridden, and confined to the house for five years. The recovery of this patient after suitable operative and corrective measures, demonstrated conclusively the paralytic basis of the condition. On the other hand, patients of a more phlegmatic temperament may complain of annoyance at first, but are able after a time to go about their usual vocations without much apparent inconvenience.

These symptoms make it particularly hazardous for some patients so afflicted to venture upon the street or in crowds. In their own homes they may be fairly free from the more severe symptoms, but on the street, where many objects are moving about, profound vertigo may develop and orientation may become difficult. It is also worthy of note that children in the adolescent period have a remarkable faculty of overcoming the symptom by the ready adaptation of which they are capable. They even learn to ignore diplopia. One boy, aged twelve years, in the author's practice, was able to play ball and officiate behind the plate without a mask, with a complete paralysis of the left inferior rectus and a partial palsy of the left superior rectus.

Vicarious Rotations of the Head.—In order to overcome diplopia and the confusion of vision resulting therefrom, the patient subconsciously assumes abnormal positions of the head. These abnormal poses were called by von Graefe "vicarious rotations of the head." The object sought is single binocular vision or vision in which confusion of images is least marked. In order to accomplish this, the patient brings the neck muscles into play. Their action is, therefore, a vicarious one.

By a careful analysis of these poses, one may divide them into two components: (1) Face rotations and (2) head tiltings. The first are truly vicarious and the second mechanically corrective. This separation is not usually made, and its omission has led to rather confused ideas as how these head poses overcome diplopia in paralytic squint.

Face Rotations.—In lateral diplopia the analysis is simple. If, for example, the right external rectus is paralyzed, the right eye is rotated inward, and the patient suffers from uncrossed diplopia, the images separating most on looking to the right and least when the eyes are turned to the left. If the face is turned to the right, or *toward the false image*, the movement is equivalent to turning the eyes to the left. In this field the eyes are in a position in which the paralyzed muscle is not called upon to act and double vision may wholly disappear. As the patient learns this fact, the face is constantly held in this position. The neck muscles relieve the eye muscles, so to speak, and diplopia is reduced to a minimum. If the right internal rectus is paralyzed the right eye deviates to the temporal side. Lateral diplopia increases on looking to the left or *toward the false image*, and diminishes on looking to the right. To overcome the diplopia the face is turned to the *left*, again *toward the false image* or *toward the field of action of the paralyzed muscle*, this being equivalent to turning the eyes to the right.

The same principles apply to the lateral rotators of the left eye. The action is a vicarious one. For lateral palsies, therefore, the following rule is established: *The face is turned toward the false image or toward the field of action of the paralyzed muscle.*

In vertical palsies the head and face postures become more complicated because: (1) Each eye has two elevator and two depressor muscles; (2) each muscle has three functions which are complementary to one another in the many positions which the eye may assume. In straight up and down movements the same rule applies as in the lateral movements. For example, if an elevator is paralyzed, the face is turned up, or the head is tilted backward. If a depressor is involved the head bends forward, or the face downward, which are equivalent. In the cardinal directions, therefore, *the face turns toward the false image or toward the field of action of the paralyzed muscle.*

Each vertically acting muscle has a three-fold function: (1) Elevation or depression; (2) internal or external rotation; (3) intorsion or extorsion. As the normal function is made up of these several components, disfunction in paralysis will similarly influence each of the components.

The analysis is best made by a concrete example: We will assume the right superior rectus is paralyzed. This muscle from "eyes front" elevates the right eye, rotates it toward the nose and intorts the vertical meridian of the cornea. When the right eye is turned outward 23 degrees, or when the muscle plane of the superior rectus coincides with the antero-posterior axis of the eyeball, elevation will be increased and its subsidiary functions, internal rotation and intorsion, will cease. In other words, all the kinetic energy of the muscle is spent in elevation. For this reason, Duane called this muscle a *right-hand elevator*. Maximum elevation is obtained when the eye is turned to the right. As this is a conjugate movement, in which both eyes share, in paralysis, failure of the right superior rectus to respond will be more pronounced when the eyes are turned up and right than when turned up and left. In other words, the vertical separation of the images will be greater on directing the gaze up and right than up and left. The face, as has been shown, is tilted upward, in the cardinal direction to overcome the vertical diplopia, and to reduce this greatest separation still more, it will be turned up and to the right. This compensatory movement brings the two eyes together, because in this position, equivalent to turning the eyes downward and to the left, the superior rectus will be least called upon to functionate in binocular movement, than in any other direction. Diplopia will, therefore, be reduced to a minimum.

If the right inferior oblique is paralyzed, instead of the superior rectus, this muscle being a left-hand elevator, the face will be tilted up and left.

The rule which is established by these two palsies, and which applies to paralysis of each vertical muscle in turn, is as follows: *The face is turned toward the normal maximum vertical pull of the paralyzed muscle.* The student will note that in the vertical palsies the phraseology is slightly different from that of lateral palsies. If in the latter it may be said, *the face turns toward the field of action of the paralyzed muscle*, the same phraseology may be applied to the elevators and depressors by inserting the words, *maximum vertical action.*

In most vertical palsies there is a moderate degree of lateral displacement. It should be noted, therefore, that the lateral tilting of the face, associated with the up or down tilting, may tend to increase the lateral separation and may, for this reason, be abandoned by the patient. In the first example, right superior rectus palsy, the eye is displaced down and out, and the vertical meridian of the cornea is tilted to the temporal side. Lateral diplopia is crossed. If the face is turned up and to the right, to relieve vertical diplopia, the right turning of the face may increase the lateral separation of the images. The recognition of this fact by the patient may tend to an absence of the lateral tilt of the face to avoid an increase in the lateral separation. As a matter of fact, the inconvenience of vertical diplopia is so much greater than that of lateral separation that every effort is made by the patient to overcome the vertical separation.

There is a third function with which the patient must reckon, namely, *torsion.* In the example used, right superior rectus paralysis, the eye is displaced down and out, and the vertical meridian of the cornea is tilted outward, because this muscle intorts the corneal meridian. According to the laws of projection and direction, if the corneal meridian is tilted outward the false image will be tilted in the opposite direction. The eye is displaced down and out, and the corneal meridian extorted, the *false image is up and in and intorted.* What does the patient do to overcome the obliquity of the false image?

Of the three phases of diplopia encountered in vertical palsy, the greatest inconvenience is suffered by reason of vertical separation. To a less extent, obliquity of the false image is a source of annoyance to some patients, and least of all, is the discomfort suffered because of the very moderate lateral separation encountered. It is well known that plus or minus cyclophoria is overcome without much subjective inconvenience in a large number of cases. On the other hand, it cannot be disputed that cyclophoria, in some individuals, is the source of much inconvenience. High degrees of oblique astigmatism which are compensated for without glasses, by action of the obliques, cause little or no inconvenience to some, but most distressing symptoms to others.

The ability to fuse in the stereoscope the images in Fig. 70 and to form a cross is well known. The horizontal lines are the same in each eye, but the vertical lines differ. In stereoscopic vision the oblique lines are observed as a vertical line as in *C*. If, however, this effort must be put forth by the hour, how many individuals will show fatigue?

It is probable, therefore, that we have grown to be too indifferent to a phase of phoria which is a source of reflex irritation to many individuals, and to this phase also of paralytic squint. The author is convinced that, although torsion of the image is more difficult to analyze than is that of lateral and vertical separation, it is a source of great distress to many patients.

Shoulder Tipping.—This brings us to *head tilting,* or what is perhaps more descriptive of the condition, *shoulder tipping*—leaning of the head to one or the other shoulder. This is a phenomenon distinct and apart from that which we have had under discussion, namely, *head facings.*

Combined with the facial posture and very occasionally alone, it will be noted that the patient's head will lean to one shoulder. This inclination of the head, in a purely mechanical way, accomplishes two things. It lowers the image on the side toward which the head is tilted, regardless as to whether the image belongs to the homolateral or contralateral eye and it corrects the obliquity of the false image. Shoulder tipping in every instance which the author has investigated is *in the direction of the physiological action of the muscle from eyes front.* It leans with the false image. Inasmuch, therefore, as it inclines with the false image and with the physiological action of the muscle, which is the same, it becomes manifestly difficult to determine exactly which symptom the patient is attempting to overcome.

In the author's studies two fundamental facts have led him to the conclusion that "shoulder tipping" is more in the interests of correcting the leaning of the image than of vertical diplopia, although it is operative in the minimizing of both conditions. They are: (1) Vertical paralysis against the rule as to lateral separation; (2) the inferiority of experimentally lowering the image by "shoulder tipping," as compared with "face rotations."

In 4 cases which the author has studied "shoulder tipping" was with the rule, and lateral separation was against the rule. In these cases it is a natural inference that the head was tilted to the shoulder to correct the leaning of the false image, the higher image being to the opposite side.

Experimentally, in testing patients suffering from vertical palsies, there is a unanimity of opinion that the "turning of the face" more effectively relieves the vertical separation than does "shoulder tipping." There is a further clinical fact to be considered, namely, "shoulder tipping" occurs relatively more frequently in hyperphoria, that is in low vertical displacements, than in vertical muscle palsies in which the separation is great. The author's conclusions from these facts are, therefore, that, although "shoulder tipping" is corrective of vertical diplopia, and of the leaning of the false image, the "turning of the face" is of greater importance in the correction of vertical diplopia than is "shoulder tipping."

Of greater importance, however, is the differentiation between "head facing" and "shoulder tipping." From a diagnostic standpoint, facing up and to the right indicates involvement of a right-hand elevator; up and left, points to a left-hand elevator. The depressor groups are equally well located by facings alone. "Shoulder tipping" is less discriminating because tipping to the right shoulder may be present in paralysis of one of four muscles, and the same number is included in tipping to the left shoulder. Facing, on the other hand, reduces the possibility to two muscles.

Summary of Symptoms.—If the student will group the symptoms discussed with reference to the chart (Fig. 29) of primary action of individual muscles, the diagnosis of the muscle involved will be much simplified.

This chart indicates the normal action of each muscle from "eyes front" or in primary position.

Symptoms which follow the chart of the normal action of the muscles from "eyes front" are:

1. False projection.
2. Position and leaning of the false image.
3. Shoulder tipping.
4. Secondary deviation of the normal eye.

Opposite to the normal action of the muscles from "eyes front" are:

1. Deviation of the paralyzed eye.
2. Decrease in the diplopia, and even fusion of the two images.

Symptoms which follow the maximum pull of the vertical muscles, or which follow the direction of the right- and left-hand elevators and depressors, are:

1. Face rotations.
2. Limitation of movements.
3. Maximum vertical separation of the images, or maximum vertical diplopia.

Associated Symptoms.—Associated symptoms which aid in the diagnosis of the character and location of lesions which may give rise to a given form of paralysis will be discussed in the section, Clinical Types of Ocular Muscle Palsies.

Diagnosis.—The diagnosis of paralysis of a muscle or a group of muscles is made by carefully studying and correlating the several symptoms and signs which have just been discussed. Primary deviation and limitation of movement are often sufficient to form fairly definite conclusions. Even though the individual muscle can be determined in this manner, the study of the case should logically proceed at least two steps further. Face and head rotations should be carefully observed, and finally, the diplopia field should be plotted and recorded.

In paralysis of the vertical muscles, primary deviation and limitation of movement may not be sufficiently definite to clearly indicate the muscle involved. Face and head rotations, if present, may be helpful, but the diplopia field always is the court of last appeal, from which the evidence furnished, is trustworthy and definite.

The question of primary or secondary deviation should be weighed in every case. Is the patient fixing with the normal eye (primary deviation), or is the patient fixing with his paralytic eye (secondary deviation)? Central visual acuity is perhaps the best guide. The patient may assist in this respect by volunteering the information that one eye has always had poor vision. If this be the case, it is probable that he will continue to fix with the good eye, even though one of its muscles may be paralyzed (secondary deviation). Furthermore, the results obtained by a study of both eyes as to limitation of movement, will definitely settle the question. The number of instances in which patients present themselves manifesting secondary deviation is small, but they occur with sufficient frequency to place the ophthalmic surgeon on his guard when the symptoms do not entirely harmonize.

SPECIAL PARALYSES.

External Rectus or Abducens Palsy.—Although paralysis of the extra-ocular muscles frequently occurs in combination, especially with other muscles supplied by the third nerve, combined palsies will be better understood by an analysis of each individual palsy separately. External rectus palsy is probably observed as an isolated condition more frequently than any other individual paralysis, save that of the levator of the upper lid, ptosis.

The muscle turns the eye to the temporal side. The eye, therefore will be displaced toward the nose. Limitation of movement will be in the direction of the paralyzed muscle, or toward the temporal side. When the affected eye fixes, under cover the normal eye will deviate inward (secondary deviation).

False Projection.—False projection will be in the direction of the action of the muscle.

Diplopia.—In paralysis of the lateral rotators, diplopia should only occur laterally. Owing to the inward deviation of the eye, the obliques gain in vertical pull what they lose in torsion of the eye. In actual practice, therefore, one may find the false image slightly higher than the true image on looking down and out, and lower than the true image on looking up and out. Furthermore, the lateral separation is apt to increase upon looking down because convergence enters into the act. Aside from these two slight modifications in the diplopia field, the diplopia will be manifest only or largely on looking to the extreme right or left. If the right eye is involved, the images will separate more and more on looking to the right, or in the direction of the paralyzed muscle. The diplopia will be uncrossed, the right-hand image traveling further to the right as the patient endeavors to turn his eyes to the right. If the left externus is involved the diplopia will increase to the left.

The face will be turned toward the side of the paralyzed muscle which will be equivalent to turning the eyes in the opposite direction, where diplopia will be reduced to a minimum or disappear.

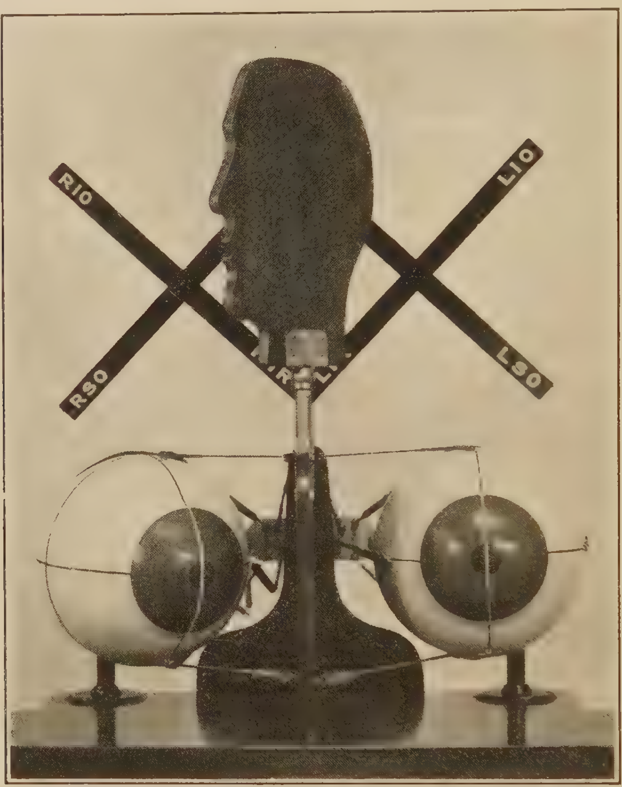


Fig. 91.—Paralysis of the right external rectus. Eye turned to the left. Face turned to the right.

Internal Rectus Paralysis.—The internal rectus rotates the eye toward the nose. In paralysis, the opposing muscles will rotate the eye to the temporal side, usually beyond the median line. *Limitation of movement* is toward the nose or toward the muscle. *False projection* is in the same direction and the corrective *face rotation* is in the direction of the action of the paralyzed muscle.

As in the case of the external rectus, diplopia should only be in a lateral direction, crossed diplopia increasing when the eye is turned toward the field of action of the paralyzed muscle or toward the false image. If the *right internal rectus* is affected, the crossed diplopia will increase to the left. As in paralysis of the external recti on looking down, the act of convergence enters into the movement and lateral separation will tend to be less in the lower field than in the upper. As Maddox[1] has pointed out, the false image of the right eye should be *lower* on looking up and in, and *higher* on looking down and in, than the true image. This is rarely observed in clinical experience, but its occurrence should be remembered by the student in order to explain the presence of vertical diplopia in the occasional instances in which it is observed.

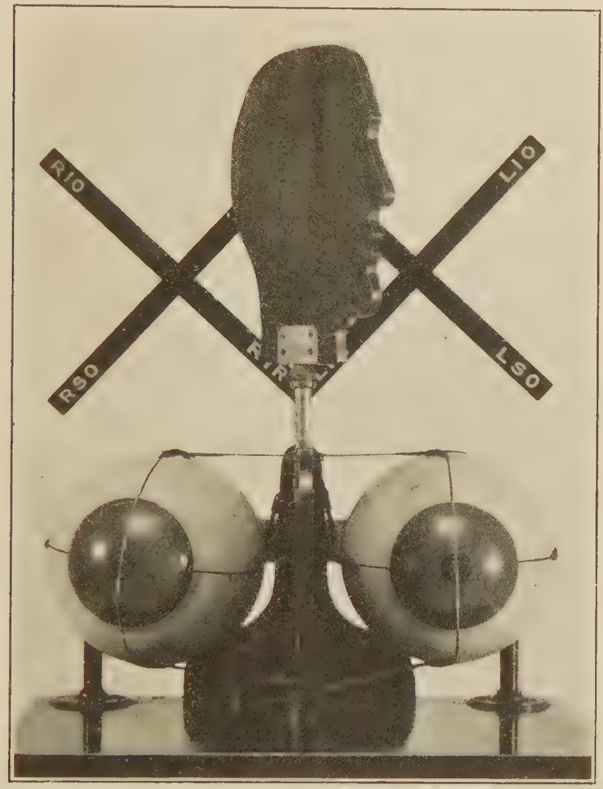


FIG. 92.—Paralysis of the right internal rectus. Eye turned to the right. Face turned to the left.

Superior Recti Paralyses.—The actions of the superior recti from "eyes front" are: (1) Elevation; (2) internal rotation; (3) intorsion. Elevation increases when the eye is rotated templeward, and intorsion and internal rotation increase when the eye is turned toward the nose.

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[1] The Ocular Muscles, page 173.

In *right superior rectus paralysis,* the eye will be displaced down and out, and the vertical meridian of the cornea will be rotated outward. Limitation of movement will be most pronounced on looking up and to the paralyzed side. False projection and the false image will be up and in, and the false image will incline toward the normal side, because the corneal meridian inclines templeward.

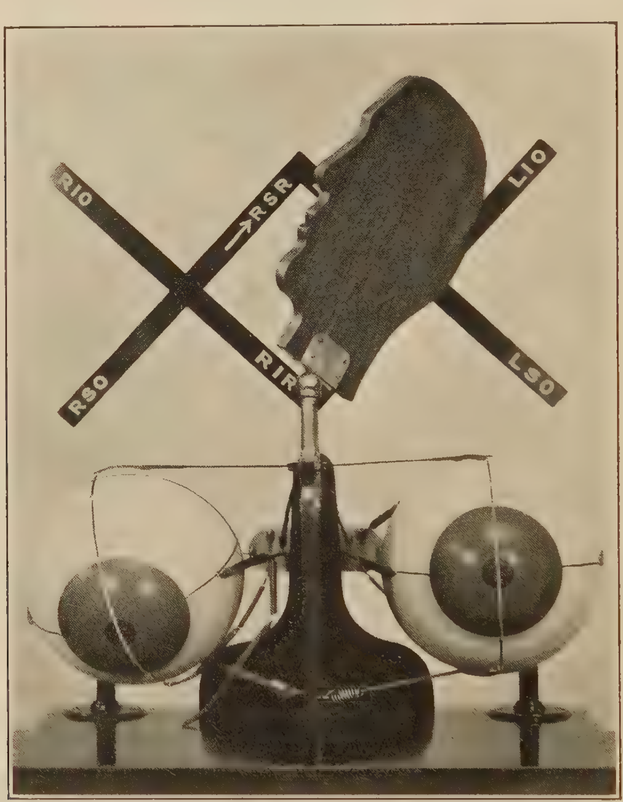

Fig. 93.—Paralysis of right superior rectus. Eye displaced down and to the right. Face turned up and to the right. Head tilted to the left shoulder.

Vertical Diplopia. Vertical diplopia increases up and toward the paralyzed side, because the right superior rectus is a right-hand elevator. There may be slight crossed separation, although this phase of diplopia is not constant.

Corrective Position of Face and Head.—The *face* is turned up and to the paralyzed side. In addition, the *head* may be tilted toward the shoulder of the normal side.

Secondary Deviation. Secondary deviation of the normal eye is up and to the normal side.

Yoke Muscle Over-action.—The yoke muscle is the left inferior oblique. If over-action of this muscle becomes apparent, the left eye will shoot up, especially when the eyes are turned to the right (Fig. 93).

Left Superior Rectus Paralysis.—*Primary Deviation.*—Down and to the left; the corneal meridian is tilted to the left.

Limitation of Movement.—Up and to the left.

False Projection.—Up and right.

False Image.—Up and right, and intorted.

Diplopia.—Vertical diplopia increases up and to the left.

Face Rotation.—The face is turned up and to the left.

Head Tilting.—The head may tilt to the right shoulder.

Secondary Deviation.—Secondary deviation of the normal eye is up and to the right.

Yoke Muscle Over-action.—The yoke muscle of the left superior rectus is the right inferior oblique. If the yoke muscle becomes over-active, it will cause the right eye to deviate upward, especially when the eyes are turned to the left.

Inferior Recti Paralyses. The inferior recti are: First, depressors; second, internal rotators; and third, extortors of the vertical meridian of the cornea. Depression increases when the eyes are rotated toward the paralyzed side; intorsion and internal rotation increase when the eyes are turned toward the normal side.

Right Inferior Rectus Paralysis.—*Primary Deviation.*—Up and out and the corneal meridian is inclined to the left.

Limitation of Movement.—Down and to the right.

False Projection.—Down and to the left.

False Image.—Down and to the left and inclined to the right.

Diplopia.—Vertical diplopia increases down and to the right.

Face Rotation.—The face is inclined down and to the right.

Head Tilting.—The head may be tilted toward the right shoulder.

Secondary Deviation.—Secondary deviation of the normal eye is down and to the left.

Yoke Muscle Over-action.—The yoke or conjugate muscle of the right inferior rectus is the left superior oblique. If this muscle becomes over-active, it will cause the left eye to deviate downward, especially when the eyes are turned to the right (Fig. 94).

Left Inferior Rectus Paralysis.—Primary Deviation.—Up and to the left, the vertical meridian of the cornea leaning to the right or intorted.

Limitation of Movement.—Down and to the left.

False Projection.—Down and to the right.

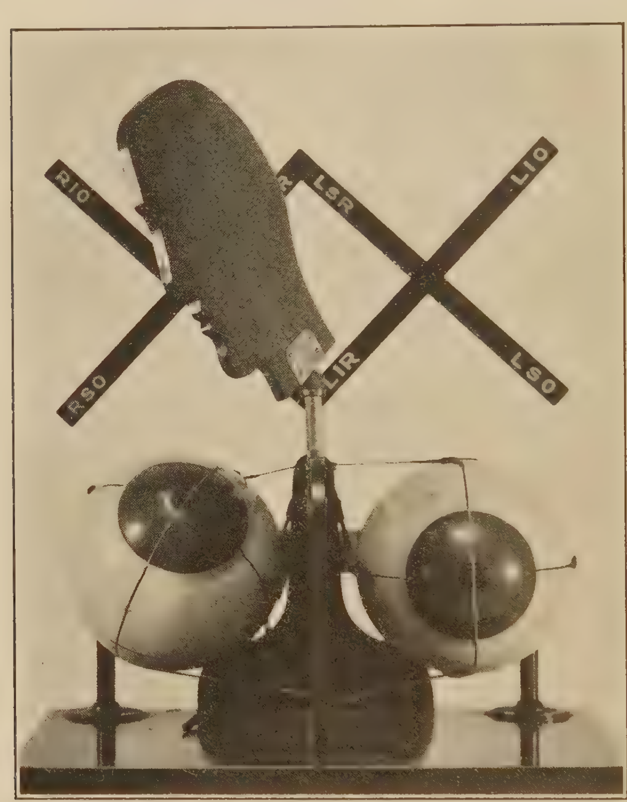

Fig. 94.—Paralysis of right inferior rectus. Eye up and in. Face down and to the right. Head tilted to the right shoulder.

False Image.—Down and to the right and inclined to the left—extorted.

Diplopia. Vertical diplopia increases down and to the left.

Face Rotation. The face is inclined downward and to the left.

Head Tilting. The head may be tilted to the left shoulder.

Secondary Deviation. Secondary deviation of the normal eye is down and to the right.

Yoke Muscle Over-action. The yoke muscle of the left inferior rectus is the right superior oblique. When this muscle becomes over-active, it causes the right eye to deviate downward, especially when the eyes are turned to the left.

Superior Oblique or Trochlear Paralyses.—The normal actions of the superior oblique muscles from "primary position" or "eyes front" are: (1) Depression; (2) outward rotation; (3) intorsion. The downward pull increases as the eye is turned toward the nose, and intorsion and external rotation increase when the eye is rotated outward. The yoke or conjugate muscle is the contralateral inferior rectus.

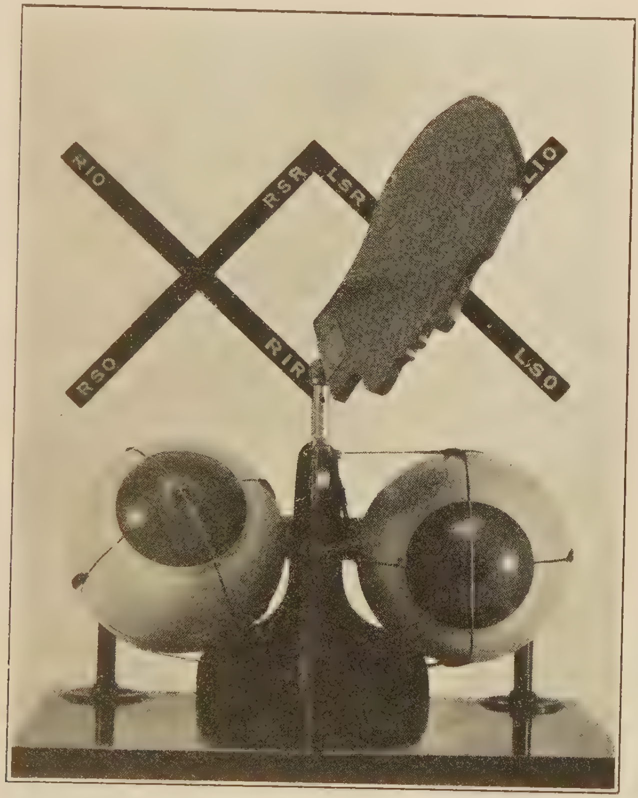

FIG. 95.—Paralysis of right superior oblique. Eye displaced up and in. Face turned down and to the left. Head tilted to the left shoulder.

Right Superior Oblique Paralysis.—*Primary Deviation.*—Upward and to the left, or normal side; the vertical corneal meridian leans to the right: extortion.

Limitation of Movement.—Down and to the left.

False Projection.—Down and to the right.

False Image.—Down and to the right; the false image tilts to the left—intorsion.

Diplopia.—Vertical separation of the images increases on looking downward and to the left.

Face Rotation.—The face is turned down and to the left.

Head Tilting.—The head may be tilted to the left shoulder.

Secondary Deviation.—Secondary deviation of the normal eye will be down and to the right.

Yoke Muscle Over-action.—If present, over-action of the left inferior rectus, or yoke muscle, will cause the left eye to deviate downward, especially when the eyes are turned to the left. This over-action also tends to modify the diplopia in the upper field (Fig. 95).

Left Superior Oblique Paralysis.—Primary Deviation.—Upward and to the right or normal side; the vertical corneal meridian leans to the left side—extorsion.

Limitation of Movement.—Down and to the right.

False Projection.—Down and to the left.

False Image.—Down and to the left, with leaning of the image to the right—intorsion.

Diplopia.—Vertical separation of the images increases on looking down and to the right.

Face Rotation.—The face is turned downward and to the right.

Head Tilting.—The head may tilt to the right shoulder.

Secondary Deviation.—Secondary deviation of the normal eye will be down and to the left.

Yoke Muscle Over-action.—If present, over-action of the right inferior rectus, or yoke muscle, will cause the right eye to shoot downward, especially when the eyes are turned to the right.

Inferior Oblique Paralyses.—The inferior obliques elevate the eyes rotate them outward, and extort the vertical corneal meridians. Elevation of each is greatest when the eye is turned toward the nose. Extorsion and external rotation increase as the eye is turned templeward.

Right Inferior Oblique Paralysis. Primary Deviation.—Downward and to the nasal side; the corneal meridian being intorted.

Limitation of Movement.—Up and to the left.

False Projection.—Upward and to the right.

False Image.—Up and to the right, with leaning of the false image to the right—extorsion.

Diplopia.—Vertical separation of the images increases up and to the left.

Face Rotation.—The face is turned up and to the left.

Head Tilting.—The head may tilt to the shoulder of the affected side.

Secondary Deviation.—Secondary deviation of the normal eye is up and to the right.

Yoke Muscle Over-action.—The yoke muscle of the fellow eye is the left superior rectus. If over-action of this muscle is present, the left eye will shoot upward, especially when the eyes are turned to the left (Fig. 96).

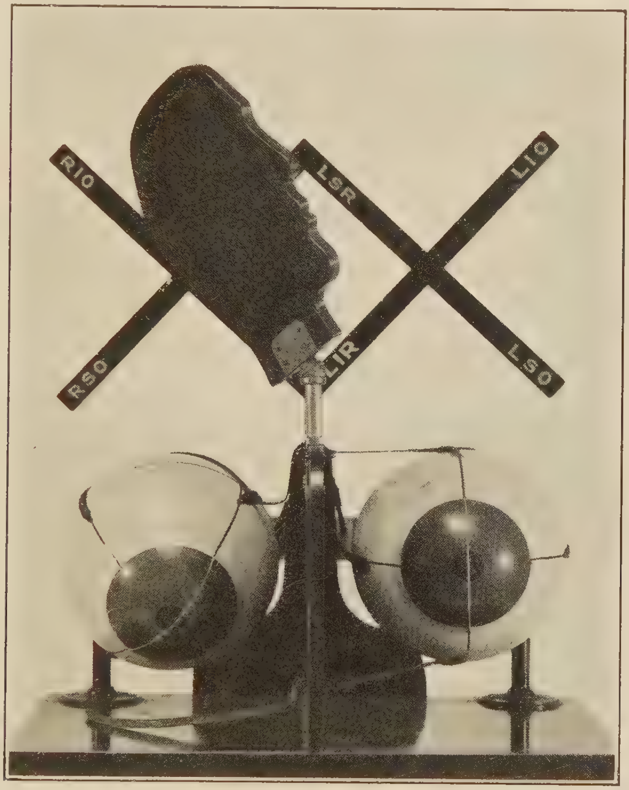

Fig. 96.—Paralysis of right inferior oblique. Eye displaced down and in. Face turned up and to the left. Head tilted to the right shoulder.

Left Inferior Oblique Paralysis.—Primary Deviation.—Downward and to the nasal side; the vertical corneal meridian will incline toward the nose—intorsion.

Limitation of Movement.—Up and to the right.

False Projection.—Up and to the left.

False Image.—Up and to the left, with leaning of the false image to the left—extorsion.

Diplopia.—Vertical separation of the images increases up and to the right.

Face Rotation.—The face turns up and to the right.

Head Tilting.—The head may tilt to the left shoulder.

Secondary Deviation.—Secondary deviation of the normal eye is up and to the left.

Yoke Muscle Over-action.—The yoke muscle is the right superior rectus. If over-action of this muscle is manifest, the right eye will shoot upward, especially when the eyes are turned to the right.

Complete Oculo-motor or Third Nerve Palsy.—Instead of isolated paralysis of the branches of the third nerve, this nerve may undergo complete paralysis. The clinical phenomena which characterize it are as follows:

Internal Ophthalmoplegia.—The pupil is dilated and unresponsive to light, convergence and accommodation, and fails to respond by consensual contraction when light is thrown upon the opposite pupil. If mydriatics are instilled, there is increased dilatation, partly from the emptying of the bloodvessels of the iris and partly through stimulation of the dilator fibers of the iris through the sympathetic system.

Paralysis of Accommodation.—Paralysis of accommodation is as complete, and behaves as though a cycloplegic were instilled. In emmetropia, central visual acuity is unaffected for distance, but the near-point can only be restored by the usual addition of a +3 diopter sphere. In hyperopia and in myopia, the correction of the total refractive error will restore normal visual acuity for distance. The near-point may be restored by the addition to the distance correction of a +3 diopter sphere.

External Ophthalmoplegia.—To the clinical symptoms enumerated in paralysis of each muscle supplied by the third nerve, is added ptosis due to paralysis of the elevator of the upper lid.

Ptosis.—Ptosis is complete, although the patient has some elevating power due to the action of the occipito-frontalis muscle. Firm pressure upon the brow of the patient, by which the action of the frontalis can be controlled, will render the patient powerless in elevating the lid. Inasmuch, however, as the sympathetic fibers which supply the muscle of Müller are intact, the instillation of cocaine into the cul-de-sac will produce a slight dimunition of the ptosis. The palpebral fissure will be widened to the extent of the sympathetic stimulation.

Primary Deviation.—The eyeball will be rotated down and out due to the action of the intact superior oblique and external rectus palsy. The vertical corneal meridian leans to the normal side (superior oblique action).

Limitation of Movement.—The only movement of which the eye is capable is that in the direction which the external rectus and superior oblique can impart to it. An attempt by the patient to rotate the eye downward will illustrate the normal action of the superior oblique combined with that of the external rectus. The eye is moved slightly down and out from its fixed position. In some instances, the torsional action of the muscle is well exemplified and the corneal meridian visibly undergoes wheel motion, the upper end being turned toward the normal side.

False Projection.—False projection is to the normal side and upward.

False Image.—False image is toward the normal side and above the true image. As the corneal meridian leans toward the normal side, the false image is extorted.

Diplopia.—Crossed diplopia occurs in the horizontal meridian and increases on looking to the normal side. Vertical diplopia also increases in the upper field and toward the normal side, the false image being extorted.

Face Rotation.—The face is turned toward the normal side and slightly upward, the position in which both lateral and vertical separation is reduced to a minimum. Face rotation and head tilting in this complex palsy are not apt to be definite in other respects than the turning of the face toward the normal side. The reasons for this are obvious. It is only with great effort that the images can be brought together and the accompanying ptosis very effectually precludes the necessity of using a cover to occlude one eye. The patient needs no encouragement to keep the lid closed. Head and face rotations are, therefore, little in evidence.

SPECIAL CLINICAL TYPES OF OCULAR MUSCLE PALSIES.

Instead of paralysis of a single muscle, there are many instances of combined ocular palsies, and in addition there are special types of ocular nerve palsies associated with other symptoms, which require at least brief consideration. Some of these symptom groups are definite and aid in the diagnosis of the character and the location of the lesions causing the palsies. Most of them fall within the domain of neurology, but it is a border line-field in which neurologist and ophthalmologist share equal responsibility in the matter of diagnosis.

Ophthalmoplegia.—When more than one muscle is paralyzed, the condition is spoken of as ophthalmoplegia.

Following the classification of the Landolts, which is a universal classification, there are four groups:

1. Unilateral partial ophthalmoplegia: paralysis of several muscle of one eye.
2. Unilateral total ophthalmoplegia: paralysis of all the muscles of one eye.
3. Bilateral partial ophthalmoplegia.
4. Bilateral total ophthalmoplegia.

Unilateral Partial Ophthalmoplegia.—A distinction should be made between *internal* and *external* ophthalmoplegia. By the former is meant paralysis of the pupillary sphincter and the ciliary muscle (accommodation paralysis). External ophthalmoplegia applies to the extra-ocular muscles, excluding, therefore, the sphincter of the pupil and the ciliary muscle.

Lesions which cause unilateral partial ophthalmoplegia may be variously located. Unless extensive, nuclear lesions are apt to cause partial paralysis. Furthermore, if extensive, there usually is involvement of some of the nuclei on the opposite side and involvement of other cranial nerve centers. Complete third nerve palsy, without involving the fourth and sixth, is of trunk origin before the nerve enters the cavernous sinus. In the latter location and after the nerve enters the orbit, a lesion causing complete paralysis of all its branches will also involve the fourth and sixth.

Partial ophthalmoplegia may be of orbital origin. It is not unusual to find several ocular nerves involved in sphenoidal and ethmoidal disease. The vagaries of trauma may bring about a similar condition. The author has under his care an instance of paralysis of the inferior and superior recti of the left eye, due to a blow from a fist, all the other muscles having miraculously escaped.

Internal ophthalmoplegia (unilateral), without involvement of the external muscles, may be nuclear or peripheral. In a majority of instances, this form of partial ophthalmoplegia is nuclear, but a sharp blow upon the eyeball may cause paresis of the sphincter of the iris and temporary loss of accommodation. Landolt[1] has observed internal ophthalmoplegia with involvement of the fourth nerve of orbital origin. He believes in these cases the cause is ethmoidal disease in a majority of instances.

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[1] Defective Ocular Movements, p. 84.

The very frequent occurrence of paralysis of the sixth nerve associated with that of the seventh in fractures of the base, or in trauma involving the base of the skull, has made this symptom valuable in diagnosing fracture of the base, when other evidence is wanting. The exposed course of this nerve renders it liable to injury in many forms of basal disease.

The Weber, Foville and Benedikt syndromes will receive special consideration (*vide infra*).

Unilateral Total Ophthalmoplegia.—Paralysis of all the muscles of one eye is found only in lesions of the cavernous sinus, in disease of the orbit, or in external trauma. A nuclear paralysis of this character cannot occur without some contralateral disturbance. Even though it were possible that a lesion could be definitely limited to one side of the floor of the fourth ventricle, so as to involve only the ocular nerve nuclei, involvement of the sixth nucleus, for example would involve the internal rectus of the other eye in conjugate paralysis to the side of the lesion, as well as disturbance of the other nerve centers or the knee of the facial nerve.

In cavernous sinus thrombosis, total unilateral ophthalmoplegia is common, but it does not remain unilateral very long. In fact, before the ophthalmoplegia on one side is complete, there usually is evidence of involvement of the muscles of the other eye.

Abscess of the orbit from disease of the nasal accessory sinus, syphilis and neoplasm of the orbit, etc., may produce a complete ophthalmoplegia limited to one side.

Trauma, such as a blow upon the eyeball and bony rim of the orbit, may cause the same condition. The author has had under his care several instances of complete unilateral ophthalmoplegia from an injury of this character. In one instance, the injury was caused by an exploding automobile tire. Recovery was tedious, but complete, with the exception of the external rectus, which remained slightly paretic after a period of six months, when the patient passed from observation. A second case was caused by a blow from a fist. In neither instance was the optic nerve involved. This, however, is not always the case. An orbital lesion which will produce paralysis of all the ocular nerves may also involve the optic nerve itself.

Bilateral Partial Ophthalmoplegia.—Lesions which involve muscles of both eyes, other than conjugate movements, as a rule are nuclear or basal in origin, although nasal accessory sinus disease must be considered as an etiological factor. The possibility also of multiple lesions occurring simultaneously should not be overlooked. In unusual types of the Gradenigo syndrome, binocular involvement has been observed. This may be due to a serous meningitis, with compression of the basilar artery as a consequence, or to coincident lesion of the sphenoidal sinus on the opposite side. A rare type of paralysis of all the extra-ocular muscles of both sides, without involvement of the intra-ocular group, is that reported by W. H. Cary.[1] This was a congenital palsy. Similar cases have been reported by Li and others.

Inflammatory disease of the base of the brain, meningitis, tumor, hemorrhage, etc., are perhaps the most common causes of these incomplete but combined palsies. Disease of the cavernous sinus has been mentioned as a cause. This rarely remains unilateral and finally may be total and bilateral.

Bilateral Total Ophthalmoplegia.—This unusual but interesting condition naturally occurs as the result of an extensive lesion of the nuclei, congenital, vascular or inflammatory disease of the base, or brain stem. In the former, the symptoms may be limited to the ocular nerves. In the latter, other cranial nerves are also involved. It is a terminal stage also of cavernous sinus thrombosis. The peculiar facial expression is known as "facies Hutchinsonia."

Ptosis.—The close relation of the superior rectus muscle and the levator of the upper lid, supplied by the same branch of the third nerve, brings this condition within the field of ocular muscle palsies. Ptosis may be congenital or acquired. As an isolated condition, it probably is the most common form of individual congenital ocular palsy, paralysis of the sixth or abducens being a close second.

The congenital variety usually is bilateral, and may appear in one or more members of the same family. Many reports have included instances of three or more generations. Associated with levator paralysis, the superior rectus is often found to be paretic. The weakness found in this muscle may in part be due to disuse because of the constantly ptosed condition of the lids. Other branches of the third nerve may be involved and the eye symptoms may be associated with other congenital defects. In some of the rarer forms, the patient can raise the eyelids by opening the mouth, a condition which is known as "jaw winking." Etiological factors found in congenital ptosis are absence or improperly developed levator muscles, adhesions and bifurcation of the muscle, and disease of the nerves or of the nuclei.

Acquired ptosis is frequently observed. It usually is unilateral. It may be caused by trauma, and by inflammatory and vascular disease at the base of the brain; it is also found in syphilis of the central nervous system, in tabes superior and inferior, in multiple sclerosis, in multiple neuritis, in hysteria, in infectious fevers such as diphtheria, influenza, in toxemia, etc. A form of ptosis is observed in swellings, inflammations and tumors of the lid. This is not a true ptosis, but to all appearances it resembles true ptosis in which the nerve is involved.

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[1] Contributions to Ophthalmic Science, dedicated to Dr. Edward Jackson, p. 122.

Recurrent Third Nerve Palsy.—Recurrent third nerve palsy, spoken of as *ophthalmoplegic migraine,* is not necessarily confined to the third nerve, but may include the fourth or sixth. The dominating symptoms are pain in the head usually followed by paralysis of one or more branches of the third nerve, with or without involvement of the fourth and sixth nerves. Palsies appear for a time and disappear, only to reappear at intervals from childhood to adult life. As a rule, the symptoms increase in severity with succeeding attacks, and finally a permanent palsy may be the result.

Pain is severe and unilateral, but it does not follow the sensory branches of the fifth nerve. It usually is of longer duration than that ordinarily found in migraine with scintillating scotoma. In most instances scintillations and other aura are absent. Pain rarely becomes generalized, but remains unilateral. It may cease spontaneously with vomiting, or it may gradually disappear. With the cessation of pain and vomiting, the usual third nerve palsy appears. This may be partial or complete. In a few instances, instead of the third nerve, the fourth or sixth have been found to be the seat of paralysis. Recurrence usually is on the same side. Cases have been reported in which one branch of the oculo-motor nerve may remain permanently paralyzed, and with recurrent attacks, the other branches become involved. The paralyses remain for a longer period than does the pain. In succeeding attacks, the palsies are often more complete and enduring. The duration varies in different cases. Attacks may only be temporary, lasting but a few days, or they may continue for several months.

The frequency of attacks also varies. In a few instances, recurrence has been observed at regular intervals, three or four times a year. In other instances, the periods of remission are longer. Mild forms which clear in four to six days have been observed as often as once a month. The more enduring types recur less frequently.

Permanent paralysis is often observed. In other instances, one or more muscles may become permanently paralyzed, while others recover.

Ophthalmoplegic migraine is regarded as a special variety of migraine. Facts which support this theory are the presence of scintillation at the beginning of an attack, and especially the fact that some genuine cases of *ophthalmoplegic migraine* begin as *scintillating migraine,* and later develop into the ophthalmoplegic variety. On the other hand, the motor variety is more enduring and more destructive. While the disease is regarded as a functional one, *an explosive neurosis,* some cases at least have an organic basis. The cause of migraine is as yet little understood, unless the theory of vascular spasm is accepted. It is probable, therefore, when we know more of the disease from an etiological standpoint, our present classification may be revised. It is possible that there is a functional and an organic variety. It is also within the range of possibilities that we are confounding two conditions. We may be including in the functional variety, because of the similarity of the two conditions, cases which are of organic origin. It is important, therefore, in the severer forms and in those cases in which permanent paralysis remains, that careful search should be made for primary organic disease of the central nervous system.

Ocular Muscle Palsy Associated with Other Symptoms.—There are a number of well known symptom groups in which paralysis of an ocular muscle becomes part of a syndrome. The following have been recognized with sufficient frequency to require mention in a monograph of this character.

Gradenigo's Syndrome.—The symptoms consist of: (1) Otogenic pathology (usually middle ear); (2) pain on the side of the face (fifth nerve sensory branches); (3) paralysis of the sixth or abducens nerve. This group of symptoms was discussed under Etiology. The prognosis as to recovery from the palsy is good, although the paralysis may continue for a period of several weeks to three months. In a few instances, recovery has taken place as late as six months after its onset. Insofar as the paralysis is concerned, operative interference upon the mastoid is not indicated. *The guide for opening or reopening the mastoid should be the otogenic symptoms.* The muscle palsy in itself is not an indication for mastoid surgery. In the author's experience, unless there is some good indication for reopening the wound, from symptoms referable to the ear, such operation does not hasten the recovery of the muscle paralysis. The ear condition and other intracranial symptoms should be the guide to the surgeon. If such indications are not present, it is wise for the ophthalmic surgeon to counsel against operative interference.

The presence of papillitis and moderate degrees of choked disk is not a part of the syndrome, but an indication of more extensive pathology than is ordinarily found. It probably indicates, according to Cushing[1] a serous meningitis. The same complication has been observed in sinus thrombosis after ligation of the internal jugular vein.

Contralateral muscle involvement usually indicates a coincident involvement of the sphenoid, contralateral otogenic disease, widespread serous meningitis (Sears), or pressure of the basilar artery in serous meningitis (Cushing).

Weber's Paralysis.—Peduncular Oculo-motor Palsy.—A lesion of the cerebral peduncle may involve the oculo-motor nerve on the homolateral side in addition to complete contralateral hemiplegia, a hemiplegia involving the face, arm and leg (Fig. 97). A, graphically illustrates the reason for this group of symptoms. The lesion is above the crossing of the face, arm and leg fibers, and the third nerve makes its exit from the brain stem at this point to the inner side of the peduncle.

Benedikt's Paralysis.—Instead of a lesion deeply seated in the peduncle, a superficial lesion in the same location may produce a third nerve palsy on the same side as the lesion, and cause irritative motor symptoms on the contralateral side of the body. This is known as Benedikt's paralysis.

Foville's Paralysis.—This group of symptoms consists of paralysis of the sixth or abducens nerve and of the facial nerve on the same side as the lesion, and contralateral involvement of the arm and leg. The lesion is situated in the lower part of the pons after the facial fibers have crossed, but before the motor fibers have crossed. The exit of the sixth nerve on the same side causes it also to suffer if the lesion is extensive enough to reach it. If the sixth nerve is not involved, the crossed paralysis is known as the Millard-Gubler syndrome. Fig. 97, *B*, graphically locates this lesion and the reasons for its appearance.

Prognosis.—The prognosis in ocular muscle palsies depends largely upon the character and location of the lesion and the underlying pathology. Congenital palsies are permanent. Birth injuries and obstetrical palsies, which are included in the congenital group, have a more hopeful outlook. Even in these cases, the prognosis is doubtful. The same rule applies as in other forms due to trauma. Much depends upon the severity of the injury.

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[1] Brain, vol. **33**, p. 204.

Traumatic Palsies.—Traumatic palsies in the acquired group are, for the most part, favorable. The sixth nerve injuries in fracture of the base, whether unilateral or bilateral, usually recover unless the injury is a direct one and the nerve is seriously damaged at the time of fracture. Ocular palsies, due to a blow upon the eyeball or bony orbit, also show a great tendency to disappear if due to pressure from blood extravasation. When due to direct injury recovery is incomplete at best. The paralysis often is permanent.

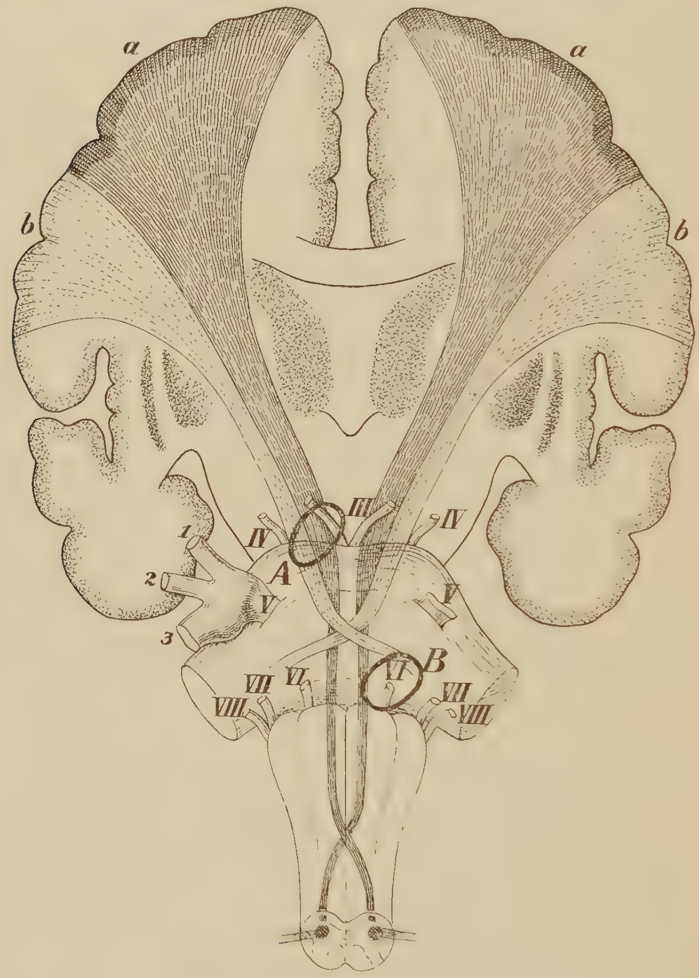

Fig. 97.—Mode of origin of alternating hemiplegia, according to Bing.

Vascular and Inflammatory Types.—A paralysis due to vascular lesions of the brain stem and the base show a marked tendency to recover. When the lesion is of an inflammatory character, such as basal meningitis from any cause or as in epidemic encephalitis, the prognosis is grave if the patient recovers from the meningitis. As stated in the section on Etiology, abducens paralysis due to otogenic pathology, Gradenigo's syndrome, disappears in a majority of instances without surgical intervention. Nuclear palsies from any cause are apt to be permanent, although in some instances they are but transitory. On the other hand, palsies due to intraorbital inflammation (abscess, sinus disease, etc.) offer a good prognosis if the cause can be removed.

In lues the prognosis is variable. As a rule, prompt luetic treatment is often followed by rapid recovery. A gummatous lesion, however, directly involving the nucleus or nerve trunk, is apt to leave a permanent paralysis. In tabes and multiple sclerosis, palsies come and go, but in many instances they become permanent.

Chronic nephritis and diabetes are the cause of many transitory palsies of the ocular muscles. It is probable that edema plays an important role in their production, but they are also toxic in origin. The prognosis as to recovery from the palsies is good.

Neoplastic Causes.—The temporary or permanent character of palsies caused by brain or orbital tumors is predicated: (1) Upon direct involvement of the nerves in the growth; (2) upon involvement from indirect pressure from the tumor; (3) upon pressure from exudate and edema. If the former condition prevails, recovery does not often occur. If the palsy is due to indirect pressure, to edema, or hemorrhage, the outlook is good.

Toxic Causes.—These are the most hopeful cases. Few toxemias leave permanent damage, whether from within the skull or peripheral. Those occurring in chronic Bright's disease and diabetes, and the peripheral palsies from diphtheria usually recover. One case of the author's of internal ophthalmoplegia, due to Vincent's angina of the mouth, left the pupillary sphincter permanently paretic. Accommodation was restored.

A form of ocular palsy which occurs in spinal anesthesia should not be omitted. The external recti occasionally become paralyzed about two weeks after the anesthesia. The prognosis is favorable. One case in the author's experience deserves special mention, as it probably belongs in this group. The paralysis was an abducens palsy in a baby which followed the withdrawal of spinal fluid. The paralysis disappeared in a few days. Inasmuch as there was nothing else uncovered to account for the palsy, which appeared promptly after withdrawal of the fluid, its presence was attributed to the spinal puncture.

Treatment of Ocular Muscle Palsies.—The patient primarily consults the oculist for relief from the subjective sensations, diplopia, vertigo, and sometimes nausea and vomiting. The first step in treatment, therefore, is occlusion of one eye by an eye pad. All symptoms immediately disappear, and rational treatment may then be instituted.

The treatment consists: (1) In the use of measures and drugs which will tend to relieve the underlying pathology; (2) in surgical procedures which will help to restore binocular single vision and correct the squint; (3) in the relief of such complications as *torticollis*, spinal curvature, and unusual poses of the head which are acquired by the patient in his own personal efforts to relieve his diplopia.

It is important in each case, if possible, to determine the character and location of the lesion. These facts not only help the surgeon to form some idea as to prognosis, but guide him in his therapeutics.

Lues is an important factor in a large number of cases, and although a positive Wassermann is not conclusive evidence of the luetic character of the palsy, it furnishes strong presumptive evidence of the same. Further confirmation should be sought by the therapeutic test, namely, the institution of antisyphilitic treatment. This, as we are all aware, has undergone radical changes during the last decade. Much advance has been made by the newer methods now in vogue. The best of the newer methods should be employed, and the patient should be given the advantage of present day antiluetic therapy. It should not be forgotten, however, that rapid mercurialization of the patient is as prompt in bringing relief in ocular and brain syphilis as are the newer methods. Furthermore, it is important that treatment should be instituted early to prevent, as far as possible, further development. To this end, the author has aways found inunctions of mercury of great value, and the best form for the administration of mercury. To this should be added the other antiluetic treatment referred to above. As luetic palsies are late lesions, iodine is of much value. The tincture of iodine should be given in 10-drop doses in milk three times daily. The dosage may be increased to the point of toleration. Inasmuch, however, as it accomplishes most good when given over long periods, a maximum dose of 15 minims three times daily is an average safe limit, which may be continued indefinitely without unpleasant symptoms. Tincture of iodine is also of great value in those cases which are due to inflammatory processes, hemorrhage, and exudate.

In a great number of cases, the treatment is that of the underlying pathology. In nephritis and diabetes, for example, elimination through skin and bowels, the correction of diet, etc., suffice. No additional measures directed toward the muscle palsy are necessary.

In those cases which are due to orbital pathology, sinus disease, etc., it is important that prompt measures be directed toward the relief of the orbital condition. Radical nasal surgery is as important in ocular muscle palsies due to this cause, as it is in disease of the optic nerve.

In otogenic pathology, the treatment must be directed to the middle ear and mastoid. It should be recalled that sixth nerve palsy in itself, is not an indication for opening the mastoid if this paralysis occurs during the course of a mastoiditis. The surgeon should be guided by other symptoms which point to radical surgery. In these conditions, however, tincture of iodine is always indicated.

Strychnia.—Strychnia has been regarded as of much value in ocular palsies. Its value is questionable, but the drug may nevertheless be used. Increasing doses are advised and should be continued for a considerable time.

Electricity.—There are differences of opinion as to the value of electricity. Faradism is popular, but its value lacks in scientific proof. When increase in muscle tone is sought, the interrupted current is of value in producing muscle contraction. The lesion, however, in most cases of muscle palsy is a nerve lesion. If electricity in any form is of value, therefore, in these conditions, it is the galvanic current to which we should look for help. This may safely be administered up to 2 milliamperes. The positive pole may be applied to the back of the neck and the negative pole to the eye. High frequency is employed by some, but there is no conclusive evidence that it is of any great value. The galvanic current is recommended.

Mechanical Measures.—Exercise of the muscle by the Michel method under cocaine has not commended itself to the thoughtful surgeon. If the nerve lesion is removed, the muscle function will be restored automatically. Better results in the exercise of the muscle may be obtained by the use of the stereoscope. This instrument tends to exercise the muscle in its proper relation to the other muscles. A single muscle never operates as an individual, but in combined action. Restoration, therefore, to normal activity is best obtained first, by removing the pathology which causes the nerve lesion; and second, by the proper exercise of the paretic muscle along with its fellows in a normal way.

Surgical Treatment.—Ocular muscle palsies, as a rule, are slow in recovering, and it is not unusual to find them present for two and three months. The question naturally arises, how long should the surgeon wait before operative measures may be safely instituted? A year after the onset of the paralysis is a safe limit, if no improvement is manifest or if such improvement has come to a standstill. In some instances, operative interference may be justified at an earlier date, but these are the exceptions.

It is a difficult matter to lay down definite rules for guidance in surgery for paralytic squint. Each case becomes a law unto itself. One can definitely state, however, that if the surgeon expects to get results by such methods as are applied to concomitant squint, he will meet with disappointment.

The most favorable form of individual muscle palsy for surgical intervention is that of paralysis of the external rectus. Advancement of the paralytic muscle and free tenotomy will not suffice to more than bring the eyes to parallelism in primary position. There will be no external rotation. The O'Connor operation offers more. This consists in shortening the external rectus and in splitting the superior and inferior recti, attaching the outer strands of each muscle to the stump of the external rectus. More correctly speaking, these detached parts of the vertical recti are attached to reflected strands of the external rectus, as shown in Fig. 98. A number of tendon transplantations have been performed by the O'Connor method, and some by more simple technic. The results are encouraging. The author has operated on 4 cases by this method, in 3 of which the results were good. In one case only an external rotation of 15 degrees was obtained. The most brilliant case was in a woman, aged forty-four years, who had a congenital palsy of the left external rectus. This patient recovered almost full rotation to the temporal side. The second case was moderately successful and the third indifferently so. In each instance, the external rectus was advanced and the internus was tenotomized and reattached well back toward the equator. O'Connor recommends local anesthesia for the operation. In the author's experience, general anesthesia is better. Paralysis of the internus may be treated in the same manner.

In paralysis of the vertically acting muscles, much may be done in a surgical way to relieve symptoms. As the displacement in this type of case is not sufficient to cause a pronounced squint, one intervenes largely to relieve the distress of the patient caused by the diplopia. Transplanting of tendons offers the best results. In the case of the superior rectus, for example, the vertical diplopia as well as the leaning of the false image can be reduced to a minimum by advancing and reattaching the tendon to a more advantageous position. A recession of the inferior rectus may be necessary to obtain maximum results. As the lower field is important to the patient, every effort should be made to restore a vertical balance insofar as possible.

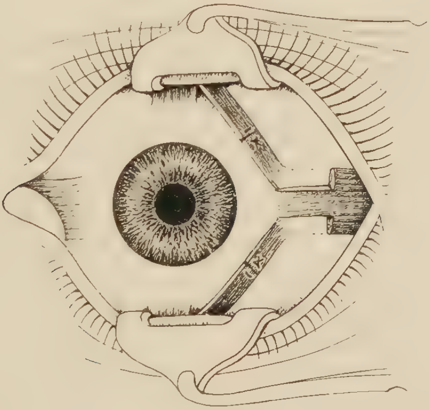

FIG. 98.—The O'Connor operation for paralysis of the external rectus muscle.

Tenotomy of an over-acting yoke muscle is of great help in reducing an imbalance to a degree which the patient can tolerate. Duane was the strongest exponent of this operation. Theoretically, it seems both logical and illogical to place the yoke muscle in the same paralytic condition as its mate. In lateral rotations, it is illogical and in practice contraindicated. Tenotomy, however, of the inferior oblique, when the superior rectus of the other eye is paralyzed, has been of wonderful aid in reducing symptoms. An unpublished case of the author serves well to illustrate its value. The case was one of paralysis of the right superior rectus. Over-action of the left inferior oblique was indicated by an up-shooting of the left eye when the eyes were turned to the right. Tenotomy of the left inferior oblique immediately relieved this symptom and brought the true and false images to a level, as tested on the operating table. After a time fatigue caused a partial return of the vertical diplopia which was relieved by advancing the right superior rectus.

Incision for tenotomy of the inferior oblique is made through the skin along the orbital rim at the junction of the inner and middle thirds. The incision is carried to the bone. A tenotomy hook passed toward the nose locates the tendon. No cutting should be attempted until the surgeon can satisfy himself, by pulling on the tendon, that the tendon is on the hook as shown by the rotation of the eye. The tendon is then freely cut with Stevens' muscle scissors.

Operation for inferior muscle palsy is even more urgent, and at the same time less satisfactory, than that of the superior muscle. An effort should be made to advance the paralytic muscle as freely as possible and to perform a recession (not a tenotomy) of the opposing rectus. In fact, the surgery should be confined to recti muscles. It is not improbable that a tenotomy of the yoke superior oblique of a paralytic inferior rectus might be as satisfactory as the same operation on the inferior oblique. It has not, however, been practised with sufficient frequency thus far to warrant an opinion as to its advisability. It offers a field for further investigation. The proper surgical attitude toward the question should be one of an open mind. If marked over-action develops, tenotomy of the muscle will probably furnish the same relief as that found in tenotomy of an over-acting inferior oblique.

If surgery cannot relieve the distressing symptoms found in paralytic squint, the surgeon has recourse to two methods. Either the paralytic eye can be occluded by means of a frosted glass, or by a bandage, or the question of enucleation may be considered. The former method is the one which usually is followed. In a few instances, at the urgent request of the patient, enucleation or one of its modifications has been practised. It is too serious for the surgeon to assume the responsibility. There are instances of extreme nervous imbalance following in the wake of an ocular palsy, which seem to justify the sacrificing of a perfectly good eye to restore nervous balance. It is a responsibility, however, which should frankly be put up to the patient for decision.

Complete third nerve palsy, if permanent, confronts the patient and surgeon with a serious problem. The accompanying ptosis precludes diplopia. From a cosmetic standpoint, surgery may be demanded. The lid may be raised by crutch glasses or by a suitable operation if enucleation is considered. It is inadvisable, however, to operate for ptosis if enucleation is not considered at the same time. Tentomy of the external rectus and the superior oblique will not remove diplopia. Muscle transplantation may bring the eye to occupy a central position in the palpebral fissure, but it will not restore rotational power. All these facts should be definitely stated to the patient before any operation should be undertaken which is purely cosmetic in character.

Measures to Relieve Complications. —The most serious complications to be met are: (1) Profound nervous upset; (2) torticollis as the result of the vicarious rotations of the head; (3) spinal curvature which is also due to the same cause.

There are extreme instances of the first complication which demand active measures for its correction. One of the most profound cases of neurasthenia which the author has ever seen was due to paralytic external and superior recti. In most instances, nervous phenomena disappear with the correction of the squint, or the elimination of diplopia. The treatment, therefore, of this condition resolves itself, into: (1) Corrective measures, either by occlusion of the paralytic eye, or the correction of the squint by operative measures; (2) treatment directed toward the profound neurasthenic condition into which the patient has drifted as the result of the paralytic phenomena. The latter is of little avail without the former, and it is almost equally urgent to follow the former corrective measures with suitable treatment, leading to a restoration of nervous and mental equilibrium. It not infrequently happens that the complications mentioned are all found in the same patient.

Torticollis is distressing. It disappears after the correction of the diplopia, but often requires prolonged treatment by massage and electricity to entirely overcome the tendency. The holding of the head in cramped positions to reduce the vertical diplopia, eventually causes spasm of the neck and spinal muscle. Occipital ache, neurasthenic symptoms and spinal curvature follow in succession. It is needless to say that until the diplopia has been removed, efforts to correct the complications will be fruitless.

Although the matter has been previously discussed, the author feels that it is *apropos* to again direct attention to the fact that "head poses" which are peculiar to certain individuals, often thought to be an individual idiosyncrasy and "assumed," should receive careful attention from the ophthalmic surgeon. They often indicate congenital or acquired ocular muscle anomalies which a careful study will reveal. The author recalls a conspicuous instance of this character selected from many others. The subject was a clergyman who was regarded as "affected" by many of his people, because he found it necessary to hold his head in a peculiar position on looking down over his congregation in order to overcome a sensation of vertigo. An examination revealed a congenital left superior rectus palsy.

The same applies to spinal curvature. Obscure cases should be carefully examined for ocular muscle disturbances, especially when the head is shown to have an unusual pose.

CONJUGATE PARALYSIS AND CONJUGATE DEVIATION.

Lesions within the brain, above the nuclear centers in the floor of the fourth ventricle, may cause binocular motor spasm and motor palsy, which are known as conjugate deviations and conjugate palsies. Lesions in and below the motor centers cause single or multiple palsies of the corresponding eye unless the lesion is large enough to include both sides. The only exception to this statement may occur in a lesion of the pons which includes the sixth nerve nucleus. The internal rectus of the opposite eye is also found to be involved in parallel movements to the side of the lesion.

Conjugate centers for movement of both eyes to the left are located in the mid-frontal convolution of the right side of the brain. Conjugate centers for movements to the right are in the left frontal convolution. A lesion in this area on the right side of the brain, if a destructive one, will cause deviation of the eyes to the right or toward the side of the lesion. The reason for this is evident. If the right frontal centers are destroyed, movements to the left cannot occur, and the opposite centers will turn the eyes to the right. This condition is known as a conjugate paralysis to the left in right-sided lesions.

If the lesion is an irritative one, the eyes will be turned to the opposite side, and the condition is known as a conjugate deviation to the left. Occasionally one finds the face is turned in the opposite direction. That is, the face may be turned to the right and the eyes to the left.

The frontal convolution, however, is not the only part of the brain in which these phenomena may occur. Conjugate movements may be disturbed by an interruption of the fibers connecting the conjugate centers with the motor centers in the fourth ventircle; by an interruption of the intercommunicating fibers, for example, in the angular gyrus which serves as a passageway for the communicating fibers. They may occur in the cerebellum because the cerebellum is a station which serves to control and govern ocular movements as well as other motility of the body.

In all these higher centers or connecting paths, the conjugate palsies follow the same laws, namely, destructive lesions cause conjugate deviation toward the side of lesion and irritative lesions give rise to conjugate deviations to the opposite side.

The exact location of the lesion is largely determined by the associated symptoms. For example, a lesion of the angular gyrus on the left side may be associated with alexia, inability to read printed or written words. A lesion in the cerebellum, associated with conjugate paralysis or deviation, may be accompanied by other symptoms of cerebellar or cerebello-pontine angle disease.

Conjugate palsies are often associated with hemiplegias. They may be well marked during the stage of unconsciousness, but gradually disappear. They may be permanent, but as a rule, are rather transitory. As Bing expresses it, "the accessory cortico-nuclear connections of the same side may gradually take over the work vicariously."

Lateral conjugate palsies also arise from lesions of the pons. In this part of the brain, *after the cortical fibers have crossed*, the eyes naturally will turn away from the lesion, if it is a destructive one and toward the lesion, if an irritative one. This, it will be observed, is directly opposite to that found in lesions of the cerebrum. Furthermore, it should be noted that a lesion of the sixth abducens nerve is associated with a corresponding disturbance of the internal rectus of the opposite side. There is a collateral connection between the nucleus of the sixth and the nucleus of the branch of the third nerve which supplies the internal rectus. This is the one exception to the rule that supranuclear lesions cause conjugate types of palsies, and nuclear and infranuclear lesions, individual palsies.

The differentiation of the location of the lesion in the brain or brain stem is made by the associated symptoms. Brain stem symptoms differ from those of the brain proper. This was discussed under Special Clinical Types of Ocular Nerve Palsies.

Conjugate Paralysis of Up and Down Movements, of Convergence and Divergence.—Paralysis of up or down movements of both eyes is rarely found alone, but usually is associated with convergence palsy. The lesion is often found along the aqueduct of Sylvius in the floor of the fourth ventricle. Not infrequently, the anterior corpora quadrigemina are involved. A very small lesion in this area will interrupt the posterior longitudinal bundles through which the fibers pass. Up and down movements apparently are not involved in lesions of the internal capsule, unless both internal capsules are simultaneously the seat of disease (Bing).

The permanence of these latter conjugate paralyses depends upon the character of the lesion. If organic or due to tumor formation, they are symptoms which often mark the beginning of the end. In cases of injury with basal involvement, the symptoms are apt to be transitory and disappear as the patient's general condition improves.

Divergency Palsy.—Divergence palsy is the rarest of all conjugate palsies. It is probably seen more frequently as a permanent symptom following lethargic encephalitis than associated with any other pathology. In its diagnosis, one must differentiate between divergence palsy and bilateral paresis of the external recti. The diplopia field is clearly differentiating. In bilateral abducens paralysis, the true and false image will separate more and more, as the light is carried to the right and to the left. In divergence paralysis, separation of the image remains the same in all parts of the horizontal field, if the same distance from the patient is maintained.

The character of the lesions which cause conjugate deviation and conjugate palsy varies. They are for the most part vascular and inflammatory, although new growths are responsible for a minority of cases. It is a symptom which rarely occurs in organic diseases such as tabes, multiple sclerosis, etc., although a limited number are on record. Injuries at the base of the brain are responsible for a fair proportion of convergence and up and down conjugate palsies. Lethargic encephalitis is a recognized cause of divergence and convergence palsy. Lateral conjugate palsies have been observed in hysteria.

Spurious Conjugate Palsy.—There are instances of spurious conjugate paralysis, such, for example, as are found in hysteria, in malingering and in cases of pain when movements are attempted in the opposite direction.

As a differentiating test between the genuine and the false von Graefe's test may be applied. The test consists in placing before the patient's eyes, two 10 or 20 diopter prisms with the apices in the direction in which movement is desired. As the patient fixes on a distant object, if the conjugate paralysis is genuine, there will be no movement of the eyes either when the prisms are placed before the eyes or when they are removed. If the eyes are capable of movement, however, a movement toward the apices may be observed when the prisms are held in front of the eyes, and a corrective movement in the opposite direction when they are removed. In malingering, this test may be of value as a means of differentiation.

Skew Deviation.—A rare condition is that known as skew deviation, a condition in which the eyes instead of deviating conjugately, may assume opposite positions. Purves Stewart,[1] records a case of extensive hemorrhage in the right lobe of the cerebellum and pons, in which the right eye was deviated down and in, and the left eye up and out. Gowers believes the lesion is in the middle cerebellar peduncle.

Duane[2] included in this group of disjunctive movements cases of anaphoria and cataphoria, *i. e.*, double *hyperphoria* and double *hypophoria*. The etiology, however, of these conditions is totally different. The cause is probably peripheral and not central, as in the case cited above. Disjunctive movements under the cover test may, however, be elicited in paralysis of one or more muscles of each eye.

Treatment.—The treatment of conjugate deviations and conjugate palsies must be directed to the underlying pathology.

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[1] The Diagnosis of Nervous Diseases, 5th ed., p. 136.

[2] Posey and Spiller: The Eye and Nervous System, p. 283.

PART V.

NYSTAGMUS OR TALANTROPIA.

RAPID oscillating movements of the eyeballs is known as *nystagmus*, or to use the term of Stevens *talantropia*. These movements vary in character, in extent, in rhythm, and in direction. They usually are bilateral, but occasionally are unilateral. The movements may be horizontal, vertical, rotatory, or mixed (combining horizontal and vertical). Bilateral nystagmus usually is conjugate in character, *i. e.*, both eyes move in the same direction, syncronously, but occasionally disjunctive oscillations are recorded. It varies in degree from coarse irregular searching movements to fine rhythmic movements of great frequency. All forms, degrees and variations are included under the same term of talantropia. All forms, like concomitant squint, cease during sleep.

Varieties.—Insofar as the oscillations are concerned, *horizontal movements* predominate. Spicer[1] found 50 per cent of the 200 cases which he examined had horizontal nystagmus. *Rotatory movements*, the eyeballs rotating around the optic axis, were obsrved in 15 per cent. In 12 per cent he found vertical oscillations. Four per cent were of the mixed type, horizontal and vertical combined. In the bilateral cases, but 1 per cent were convergent, all others were parallel in character. Unilateral cases, excepting in spasmus nutans of children, are rare. Unilateral cases, as a rule, are vertical in character. The oscillations are very rapid and of short amplitude.

Character and Frequency of the Oscillations.—Maddox estimates the frequency of the movements to be from 10 to 350 per minute. The extent or amplitude varies from oscillations, almost imperceptible to the naked eye, to 5 and, in some instances, even more millimeters. Nystagmus which dates from early childhood and vertical nystagmus of spasmus nutans is rapid. Most cases which are due to acquired defects in vision are slower and coarser than those which occur in congenital cases. The movements observed during convalescence from a paretic muscle, when the eyes are turned as far as possible in the direction of the paralyzed muscle, are coarse and jerky, lacking in the rhythm usually observed in other forms. This type of nystagmus is recognized under the term pseudonystagmus by some authors.

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[1] Ophthal. Rev., May, 1914.

Coppez[1] recognizes the oscillations first as spring nystagmus, and second, as undulatory. In the first, there is a slow movement followed by a rapid recoil. In the second, the movements are equal. A typical instance of the first is that of vestibular origin. The second type is observed in miner's nystagmus. Although Coppez discusses the underlying pathology of both forms at length, no explanation, acceptable to all, has thus far been produced to mechanically explain the reason for the difference in the character of the oscillations.

Methods of Studying the Oscillations.—As Edward Jackson[2] pointed out, there is no difficulty in studying and measuring the amplitude when the movements are slow and coarse. He suggests the holding of a ruler before the eye to measure an amplitude of 2 mm. or over. For the same study, the corneal reflex from an electric light may be observed. Rapid movements of slight amplitude require an instrument with some magnifying power, such as the ophthalmoscope.

Wilson[3] offers an ingenious device for the same purpose. For proposes of magnification, he places 20 diopter spheres in a trial frame before the patient's eyes. Two 12 diopter prisms are held apex to apex and in such a position that the two corneæ are brought together so that they touch. This offers an advantageous position to study the movements of the two eyes together.

The ophthalmoscope focused upon a retinal vessel detects the presence of fine nystagmoid movements, which may not be recognized by the naked eye.

Study by Slit Lamp and Ferree-Rand Perimeter.—Two of the newer instruments now in use offer a better means of studying the character and extent of the oscillations than any of the methods thus far devised, or at least available in an average office. They are the slit lamp and the Ferree-Rand perimeter.

In the former, the F 55 objective with a micrometer ocular not only enables the observer to study minutely the character of the movement, but the micrometer ocular can be used to advantage in measuring the amplitude of the oscillation. A fine adjustment of the band of light can be made on the pupillary edge as it moves along the scale in the ocular, and deductions can be made on the amplitude according to the rules laid down for the estimation of dimensions of any definite area.

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[1] Arch. d'ophthal., September, 1913.

[2] Ophthal. Rev., January, 1910.

[3] Brit. Med. Jour., November 22, 1913.

On the Ferree-Rand perimeter, the ophthalmoscopic centering device is so definite and refined that similar observations can be made on this instrument. Although a micrometer scale in its present form is not a part of the equipment, such an addition could be made for purposes of measuring the amplitude of oscillations.

The advantage of these two methods, aside from accuracy and the ease with which studies can be made, is the fact that no additional instrument need be added to one's equipment to meet the emergency for this special study.

Etiology.—It is difficult to classify all forms of nystagmus from an etiological standpoint. This difficulty arises from the great number of conditions in which nystagmus is found, and also in a great measure, from a lack of thorough understanding as to how the oscillation is brought about.

The classification of Maddox is probably as simple and at the same time as comprehensive as any. In the first group, he includes visual defects; second, neuro-motor exhaustion; third, organic diseases.

Visual Defects.—Visual defects are the predominating factors in early childhood. If these defects are present before fixation has been acquired, even though the visual disturbance may disappear, as Maddox points out, nystagmus may remain. This fact explains many cases of nystagmus in older children, which cannot otherwise be explained even though there may also persist moderate lowering of central visual acuity. Among the causes operative in childhood, congenital cataract, corneal nebula from ophthalmia neonatorum and macular disease are the chief.[1] It is probable, however, that visual disturbances such as high degrees of astigmatism which are not corrected at this early period are also responsible for a number of cases. The author has observed a number of cases of this type for which no other causes could be found.

In adults, visual defects do not produce nystagmus unless macular vision is disturbed. It is observed in complete monocular blindness, but here again macular vision is also lost. In cases of squint in which nystagmus sometimes is observed, it is probable that in each instance, either congenital amblyopia or amblyopia exanopsia is present.

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[1] Labyrinthian nystagmus is not included in the forms discussed. For a study of this form, the reader is referred to the many excellent books on the ear and monographs on labyrinthian disease.

Neuro-motor Exhaustion.—Neuro-motor exhaustion probably expresses exactly the cause of miner's nystagmus. The cramped position in which the miner works, associated with poor illumination, brings about the condition. When these patients are rescued from their usual occupation and are given fresh air and light, the nystagmus disappears. Like other forms of occupational neuroses, the oscillation is apt to recur if the occupation is resumed.

If *neuro-motor exhaustion* is broadly interpreted, nystagmus associated with the nodding spasm of infants may be said to be due to this cause. It occurs in children suffering from rickets. The nystagmus is more often unilateral than bilateral. It is, as a rule, vertical in character and very rapid. Accompanying it, is the characteristic nodding of the head. In the author's experience, the head nodding is in the vertical direction, although he has seen a number of cases of side to side movements of the head. The underlying factor is malnutrition and with the correction of the rickets, nystagmus and head nodding disappear. The prognosis, therefore, is good.

There is a form of lateral nystagmus and lateral head movement which persists through childhood and even into adult life. It is probable that some of these cases, while not the typical nodding spasm just referred to, also have a basis of malnutrition very early in life. Although the body nutrition is corrected, the habit remains. Such forms of nystagmus are more or less intermittent. There are times when all movements cease for a few moments, but recur especially when the child is under tension. These children are of the "nervous" type, timid, and retiring, and extremely sensitive. They rarely recover completely either from nystagmus or from head rotation.

Organic Disease. Nystagmus may occur in disease of any part of the central nervous system which will disturb the function of the conjugate centers. Middle ear disease, lesions of the pons, anterior quadrigeminal bodies, optic thalami, cerebellum and peduncles are especially apt to be accompanied by nystagmus. It is a characteristic symptom of Friedreich's disease, a symptom which distinguishes it from locomotor ataxia. In this disease, the nystagmus consists of an irregular oscillation of the eyeballs when attention is fixed upon an object moving especially in the horizontal plane. The symptom is rarely absent in Friedreich's disease, and if absent, can be elicited by causing fixation on a moving object, or by turning the patient around his vertical axis. It is not within the domain of ophthalmology to differentiate this disease from hereditary cerebellar ataxia, excepting insofar as the latter disease is associated with many eye symptoms such as optic neuritis, ocular palsies, etc., whereas nystagmus may be the only eye symptom found in Friedreich's disease. As to location, the pathology of Friedreich's disease is supposed to be located in the cord; that of hereditary cerebellar ataxia, in the cerebellum.

Nystagmus has not been observed in paralysis agitans, a condition in which one might expect to find it. The reasons for its absence are to be found in the fact that lesions of paralysis agitans rarely involve the cerebellar pathways.

In multiple sclerosis, oscillation of the eyeballs is a characteristic symptom. It is similar in character to that found in Friedreich's disease. When at rest, there is no evidence of nystagmus, but fixation especially along the horizontal plane, causes it to appear. Like that of Friedreich's disease, it can also be elicited in fixation in the horizontal plane. It is not a true rhythmic oscillation, but somewhat irregular. Because of the underlying pathology, no disease of the central nervous system is more varied in its symptomatology and insofar as eye symptoms are concerned, nystagmus is perhaps the most constant and characteristic when present.

According to Gowers, nystagmus is occasionally found in spastic paraplegia, in ataxic paraplegia, in extreme cases of multiple neuritis due to profound toxemia, in progressive muscular atrophy, and in syringomyelia.

Disease of the subcortical areas of the cerebrum is rarely accompanied by nystagmus, excepting in such conditions which arise from wide-spread toxemias, either from within or from without. The cerebellum, on the other hand, being a controlling center, through which associated fibers must pass, when disturbed by pathological processes, is apt to give rise to nystagmus. In disease of the lateral lobe, oscillation of the eyeballs usually develops when the patient looks toward the side homolateral to the lesion.

Treatment of Nystagmus.—In those cases which are due to central visual disturbances, treatment must be directed to the cause. The development of this complication in congenital cataract is an argument in favor of early operation in this condition. It is not the only consideration which should determine the time of operation, but it is an important one. Even though central visual disturbance is corrected, if nystagmus has been present for a considerable period, it may remain throughout life.

If no pathology of the media is found in nystagmus of young children, and no evidence of macular disease can be uncovered, high refractive errors, especially if astigmatic, should receive careful attention on the part of the physician. In albinism, little can be done, but the constant wearing of dark glasses may be of some aid.

In the second group of causes, neuro-motor exhaustion, the prognosis is good if the cause is removed. Miner's nystagmus yields to treatment if the patient is removed from his usual occupation, brought into good light, and treated with tonics.

Nystagmus of infants, associated with spasmus nutans, yields to treatment directed to the rachitic condition which is the underlying cause. The prognosis in these cases is always good.

It is evident that in a majority of instances in which the nystagmus is due to organic disease, no improvement can be expected, unless the underlying pathology can be removed.

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